The Journal

of the

Louisiana State Medical Society

Vol. 106, No. 5
$4.00 Per Annum, 35c Per Copy

MAY, 1954

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

THE COMMUNITY POLIO CENTER

J. D. MARTIN, M., D.

BATON ROUGE

At the time I was asked to prepare this paper the topic was pertinent. Advances in medical knowledge and technology in the past year suggest that in the near future, if we apply this knowledge, that polio like diphtheria and smallpox will no longer be a major problem causing a need for polio centers. At the present, however, statistical evidence still points up a need for polio centers.

For the nation since 1915, with the exception of 1916, there has been a steady increase in the number of cases of infantile paralysis and a steady decrease in the number of deaths per 100 reported cases, but almost no change in the death rate per 100,000 population. (Table 1).

Excluding 1952, for which figures were not available while preparing this paper, the highest death rate per 100,000 population was in 1916 when the rate was 9.4 or five times what it was in the second worse year, 1927, with a rate of 1.9. (Table 1)

The highest death rate per 100 reported cases was 42.4 in 1918. The lowest was in 1951 when it was 4.7 per cent. (Table 1).

The five years in order, from greatest to least of the five, in which the greatest number of cases of polio have been reported since 1915 are 1952 (57,628), 1949 (42,033), 1950 (33,303), 1951 (28,395), 1948 (27,726). (Table 1).

TABLE 1.
CASE, DEATH AND FATALITY RATES—POLIOMYELITIS—UNITED STATES—BY YEAR *

Year	Cases Per 100,000 Pop. †	Deaths Per 100,000 Pop. †	Deaths Per 100 Reported Cases
1915	3.1	1.0	40.3
1916	41.4	9.4	26.2
1917	5.0	1.5	34.8
1918	2.9	1.1	42.4
1919	2.3	.8	41.3
1920	2.8	.8	36.8
1921	6.9	1.7	29.7
1922	2.4	.8	38.1
1923	3.4	.9	30.6
1924	5.6	1.0	22.0
1925	5.5	1.4	27.5
1926	2.5	.8	36.1
1927	8.8	1.9	20.8
1928	4.7	1.2	28.2
1929	2.7	.7	30.1
1930	7.9	1.2	15.5
1931	14.6	1.7	13.6
1932	3.2	.7	23.4
1933	4.3	.6	16.0
1934	6.0	.7	11.3
1935	8.5	.8	9.6
1936	3.5	.6	17.3
1937	7.4	1.1	15.4
1938	1.3	.4	28.6
1939	5.6	.6	10.5
1940	7.4	.8	10.4
1941	6.8	.6	8.9
1942	3.0	.4	13.9
1943	9.3	.9	9.2
1944	14.3	1.0	7.2
1945	10.3	.9	8.7
1946	18.4	1.3	7.2
1947	7.6	.4	5.4
1948	19.0	1.3	6.8
1949	28.3	1.8	6.5
1950	22.0	1.1	5.1
1951	18.8	.9 ‡	4.7
1952	37.0	N. A.	N. A.

* Statistical Services Dept. N. F. I. P.—1-53.
† Based on Population of States Reporting
‡ Based on 10% Sample of Death Certificates
N. A.—Not available
Note: Morbidity data for earlier years should be interpreted with caution because of probable underreporting of less serious cases.

* Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

The five years in order, from greatest to least of the five, in which the highest rate per 100,000 population has been reported are 1916 (41.4), 1952 (37.0), 1949 (28.3), 1950 (22.0), 1948 (19.0) (Table 1)

For Louisiana the number of cases of polio per 100,000 population per year with corresponding rate since 1948 are: 163 (6.3), 227 (8.6), 409 (15.2), 836 (31.1), 863 (33.0). (Table 2).

TABLE 2.
ANNUAL NUMBER OF CASES OF POLIOMYELITIS REPORTED AND CASE RATE PER 100,000 POPULATION—LOUISIANA—1948-1952 *

Year	1948	1949	1950	1951 †	1952 †
Cases	163	227	409	836	863
Rate	6.3	8.6	15.2	31.1	33.0

* Statistical Services Department—National Foundation for Infantile Paralysis—Source—United States Public Health Service
† Provisional

Louisiana from 1948 to 1952 has had an annual average of 500 cases to rank it 25th in the nation for number of cases. (Table 3). In 1952, Louisiana reported 863 cases to jump it from 25th to 19th in rank in the nation in number of cases reported.

TABLE 3 *
NUMBER OF CASES OF POLIOMYELITIS REPORTED; CASE RATE PER 100,000 POPULATION † AND RANK AMONG STATES FOR NUMBER OF CASES REPORTED AND RATE—LOUISIANA FIVE YEAR AVERAGE 1948-1952 ‡ AND 1952 ‡

Number of Cases				Cases Rate Per 100,000 Population			
1948-1952		1952		1948-1952		1952	
Annual Avg.	Rank	Annual Total	Rank	Annual Avg.	Rank	Annual Rate	Rank
500	25	863	19	19.1	33	33.0	28

* Statistical Services Dept.—N. F. I. P.—Source—United States Public Health Service.
† Based on April 1, 1950 Population for 5 year averages and for 1952 State Date; estimated July 1, 1952. Population for 1952 U. S. Total.
‡ Data for 1951 and 1952 are provisional.

The case rate per 100,000 population for Louisiana for the five year period, 1948 to 1952, averaged 19.1 to rank Louisiana 33rd in order of frequency of the States of the nation. The case rate in 1952 was 33.0 as Louisiana jumped from 33rd to 28th place among the states. (Table 3.)

In 1951 and 1952, polio occurred in almost every parish in Louisiana. For the past five years polio has been steadily increasing each year in numbers of persons infected with the polio virus. At the present time there are four polio centers operating in Louisiana. These are located in the Charity Hospital, New Orleans, the Baton Rouge General Hospital, Baton Rouge, the Baptist Hospital, Alexandria, and the Charity Hospital, Shreveport.

These four centers admitted and treated 785 of the 863 persons reported as infected with polio virus in Louisiana in 1952. The remaining number of cases were treated outside these centers.

Only three parishes in 1952, Madison, Tensas, and West Feliciana, did not send patients for care to one of Louisiana's four treatment centers. East Baton Rouge Parish sent the greatest number of cases, 161; while Orleans Parish was second with 101. Caddo was third with 30 cases and Rapides and Ouachita each sent 28 cases to a treatment center.

In addition to having the greatest number of cases in 1952, East Baton Rouge had the highest incidence with one case per 1,000 population. In 1952, there was a shift of area of highest incidence from north Louisiana, in 1951, to south Louisiana, in 1952.

Of the 785 persons treated in the centers in 1952, 560 were 9 years of age and under and 110 were over 20 years of age; 664 were white and 121 were negroes; 439 were males and 346 were females. The youngest person admitted for treatment was a five week old boy and the eldest was a 49 year old white woman (Table 4).

TABLE 4
DISTRIBUTION BY AGE, COLOR AND SEX OF PATIENTS ADMITTED TO LOUISIANA'S POLIO CENTERS—1952

Ages	White		Negro		Total For ages
	Male	Female	Male	Female	
Under 1 yr.	21	25	5	1	52
1 - 4	129	98	36	25	288
5 - 9	116	78	16	10	220
10 - 14	39	25	14	2	80
15 - 19	12	17	4	2	35
20 - 29	31	47	3	1	82
30 - 39	11	10	0	2	23
40 & Over	2	3	0	0	5
TOTALS	361	303	78	43	785

One person died of polio in 1951, and 17 in 1952. Louisiana's polio mortality rate in 1951-1952 was slightly more than 2 per cent as compared to a reported national average of 6 per cent. Twelve of the 18 persons dying were females and 17 were white. Eleven of the deaths occurred in persons past 21 years of age. Of these 11, 9 were women. In 7 deaths under 21 years of age 1 was a teenager and 6 were 8 years of age or younger. Four of the 7 were male and 3 were female; 6 were white and one was negro.[2]

One or more cases of polio occurred each week of 1952 with July 20-26 being the worst week, with 50 cases reported. One-hundred and eighty-five cases in July made July the worst month of the year. Fewer cases (11) occurred in November than in any other month.[2]

Of the 785 persons admitted to the polio centers of the state in 1952, 698 were discharged after an average hospital stay of approximately forty days each. Eighty-six persons admitted to the centers in 1952, were still there on January 1, 1953. As of that date these had an average hospital stay of approximately 118 days each.[2]

This statistical data has been presented for the specific purpose of suggesting that the diagnosis and care of persons infected with polio virus is becoming increasingly important because of the increasing number of cases occurring in this country, that expanding medical knowledge and skills relating to the person infected with polio virus require a specialized facility to integrate the men, machines, money and skills into a unified attack against polio and to demonstrate some of the problems facing these polio centers from the standpoint of housing persons of different sexes, age groups, etc.

Although polio is worldwide in its occurrence, paralytic polio is more frequent in the temperate than any other zone, so we who live in the temperate zone must be constantly alert and ready to meet this challenge.

Polio occurs most often in the temperate zone between the months of July and November, but it also occurs in every other month of the year. This, plus the fact that persons infected with polio virus may require extended hospitalization, necessitate providing in the center an adequate number of trained personnel and beds and sufficient equipment to meet the community's usual needs for polio and to permit adequate expansion in times of unusual incidence.

Having considered the reasons for having polio centers and some of the problems associated with housing in a polio center, it is well at this time to define what is meant by "Polio Center." A polio center is a facility serving a large or small geographical area, depending upon the population density, and which may or may not cut across governmental or political subdivision lines.

The center is the brain in the community's fight against polio. Here are integrated the knowledge, skills, equipment, methods, drugs and money made available by the community to fight polio.

There is no blueprint for the organization and operation of a center. Each community must resolve the problem in its own way. There are certain principles which will serve as guideposts. Wise application of these principles adapted to the local situation will result in a well planned, well operated center, meeting a real community need.

Essential to the development and operation of a polio center is widespread individual and group participation at the community level. The development and operation of a center requires careful planning geared to the communities resources and the needs of the individual patients who will use it, and their families, and this is best studied and worked out by many persons working as team rather than by one expert.

Adequate housing for sex, age group, and status of the disease must be provided. This means rooms for isolation of patients in the acute phase of polio, and for patients with polio who may be also infected with other communicable disease, adequate

space for special-care patients such as those in respirators, needing oxygen, post-tracheotomy care, rocking bed routine and special observation during the period of toxemia and of bulbar and intercostal paralysis and space for the postacute cases according to age group and sex.

Inseparable from adequate housing for the adequate care of persons sick with polio are competent medical, nursing and ancillary personnel, such as physiotherapists, occupational therapists, laboratory technicians, and mechanics of the skill trades, together with the tools these folks need to properly perform their function.

The patients for whom the Center serves are entitled to care regardless of the individual's personal ability to pay for the needed services. Each person, however, should be expected to pay for his care according to his ability. This may be from his personal means or through insurance which has been purchased for this purpose. The person's ability to pay is exceeded when he has to start mortgaging real property or when he has to borrow to start paying for the services he is receiving. At the time that this point is reached the National Foundation for Infantile Paralysis should assist the patient in meeting his financial obligations. Since the money for this purpose is donated annually by folks all over the country through the March of Dimes, any person afflicted with polio should expect and receive this aid without considering it a handout. Because any one of us may some day stand in need of this help each of us should generously support the March of Dimes and in addition obtain insurance which will help us foot the bill if one is presented to us. This is important because we can expect adequate services from our centers only if they are adequately financed and if we do not support them voluntarily we may have to support them through taxation.

Once a center is established, housed, staffed and equipped the real job, the job of operating the center, begins. No matter how well housed and equipped a center is, it is only as good as the men and women who staff and use it. Good operation of a center is based on the team concept. The team is made up of patients, doctors, nurses, physiotherapists, occupational therapists, dieticians, laboratory workers, skilled mechanics, aides, porters, maids, druggists, community volunteers, the families and friends of patients, the local chapter of the National Foundation for Infantile Paralysis, the insurance companies, who write polio insurance, the official health agency of the community, and the hospital administrator. Each of these has an interest in the center and certain responsibilities to the center. Because each is a member of the team, each should be represented on the governing body of the center. This body should be given the responsibility and authority for defining the responsibilities of each of the groups serving the patient through the team. This requires study, discussion, planning, and operation for the good of all. Agreements should be reached and established as policies. To be effective these policies should be broad, flexible, written, and subject to review not only when the situation indicates a change, but subject to periodic study and analysis. Policies based on sound principles should govern every sphere of activity of the center.

In the organizational set-up of the center special attention should be given to the selection of a well trained medical administrator noted for his ability to get along with people. In this individual should be vested the authority to implement the policies established by the governing body. Working in close harmony with the administrator should be an advisory committee of professional personnel representing the different specialty medical and technical groups servicing the center. This committee should meet with the administrator to consider all technical problems and give the administrator the benefit of their thinking so that he may properly advise the Board to help them formulate policies governing technical matters so that he may better establish sound practices and procedures in the

technical field of operation of the center.

Maintenance and improvement of knowledge and skills of technical people is needed if the center is to operate most effectively. To this end in-service training programs and attendance at institutes, workshops and courses of formal instruction are necessary. Opportunity to attend such instruction should, therefore, be made available to all full time professional and technical personnel of the center. If the center provides its personnel with generous salaries these folks can then be expected to pay their own way to these educational programs. If, however, the salaries paid are only average for the community in which the center is located and are not adequate for the worker to obtain the training without real hardship, it is well to remember that the benefit from the training accrues to the community and the center should provide in its budget for training these workers.

The effectiveness and problems of the center can only be measured by the results it achieves or fails to achieve. These results can be measured only if they are recorded in such manner that the recorded information lends itself readily to sorting and analysis. Most hospitals and centers are still in the horse and buggy days in this regard. The most costly item in any budget today is salaries. Hand tabulation and sorting uses many hours of a worker's time when the same job could be done in a matter of minutes if the recording, sorting and tabulation of data were done by business machines designed for that purpose. Utilization of suitable machines in the centers would result in more accurate information early enough for it to be of value in planning for the future.

Two special problems which each center must face and solve are "who is to be responsible for the medical management of the patient sick with polio?" and "what is to be done to handle the emergency that arises when the medical person responsible for the patient is out of pocket?"

Obviously, the physician of the patient's choice is the one responsible for the medical management of the case. Expanding medical knowledge, however, has made it difficult for any physician to be thoroughly competent in several different specialties. To assist the family physician when he wants advice and to care for those patients admitted to the center who do not have a physician of their own, the center should establish a team of specialists—an orthopedist, pediatrician, internist, neurologist, otolaryngologist, physical medicine man, pathologist, public health physician, physiotherapist, and a nurse specially trained in the nursing care of persons with polio. This team should function as a team with all persons on whom it is called in to help. Only with close integration of knowledge and skills of these specialists working in harmony with all other persons in the center can the patient receive optimum care.

To properly handle the emergency which arises while the attending physician is unavailable the nurse should have written standing orders. These orders should be agreed on by all medical personnel using the center, should be posted in conspicuous places where they will be readily available in time of need and should be clear and detailed enough so that there is no doubt in the nurse's mind about what she should do until the attending physician can be located.

There are other problems associated with the operation of centers which the governing board of each center will recognize. If the board first defines the principle which should govern the solution of the problem then applies the principle by the one best method for the achievement of the desired end each center will satisfactorily work out its own difficulties.

In conclusion, I hope that I have demonstrated that the need for community polio centers continues until vaccination against polio becomes an accepted, effective preventive measure. I wish to stress that application of the principles of good human relations and administration are essential to the proper operation of a polio center. A recommendation also seems in order. It

is fairly well established that fatigue aggravates the manifestations of polio yet it is necessary for some patients to be transported such long distances in Louisiana to reach a polio center that they are subject to considerable fatigue. To offset this adverse factor consideration should be given to the establishment and operation of polio centers in Monroe and either in Lafayette or Lake Charles.

REFERENCES

1. Poliomyelitis—1952. Annual Statistical Review—Statistical Services Department, The National Foundation for Infantile Paralysis, Inc. January 1953.

2. Polio—Louisiana Treatment Centers—1952. Louisiana Chapter of the National Foundation for Infantile Paralysis.

—o—

CHRONIC DISTENTION OF THE URINARY BLADDER; ITS CLINICAL AND THERAPEUTIC SIGNIFICANCE *

C. D. CREEVY, M. D.†
MINNEAPOLIS, MINN.

Since the Ebers papyrus, physicians have regarded sudden emptying of the chronically distended bladder as dangerous. While this is supported by most urological textbooks, there is no evidence that it is more than a supersition. To understand how this developed one need only remember that, before modern prostatic surgery attained its low mortality, treatment of the obstructing prostate was postponed until discomfort became intolerable. The resulting vesical distention caused hypertrophy and later atrophy of the detrusor, with the formation of cellules and diverticula and led in turn to dilatation of the upper urinary tract; uremia with imbalances of water and electrolytes commonly followed. The resultant anorexia led to hypoproteinemia and vitamin deficiencies, while the patient's efforts to diminish his frequency of urination naturally caused dehydration. That any additional disturbance of the urinary tract in such a patient should lead to disastrous reactions is now easily understood, but theories concerning the noxious effects of sudden emptying of the bladder were formulated in ignorance of the factors just mentioned, and even of bacteriology. Survival of these ideas is a tribute to the authority of the printed word. The literature explains the dangers of sudden decompression of the bladder as follows:

I. Lesions of the urethra and bladder
  A. Direct trauma
    1. Hemorrhage (Finlayson 1884).
    2. Absorption of urine through traumatized area (Sedillot 1861)
  B. Indirect trauma
    1. Hemorrhage ex vacuo (Albarran (1883)
    2. Laceration of engorged vesical veins by spastic detrusor (Mercier 1861)

II. Abrupt release of intrarenal pressure.
  A. Congestion (d'Etiolles 1876)
  B. Edema (Regnault 1883)
  C. Distortion of renal tubules (Wright 1919)

III. Reflex nervous disturbances
  A. Syncope (Guyon 1879)
  B. Sudden hypotension (Pilcher 1914)

While many authors maintain that sudden decompression of the bladder is risky, they present no proof that the *rate* of emptying is important. No complete autopsy protocol is to be found, nor have the exact circumstances presumed to make abrupt decompression dangerous been determined. There is no evidence that rapid removal of the urine causes lesions differing from those seen after gradual emptying or in the untreated patient dying of prostatism, nor has anyone explained why amounts of urine as large as 14,000 cubic centimeters have been removed abruptly without ill effects. Other objections to the theories outlined are as follows:

The trauma of catheterization does cause hematuria, but the patients who

* Presented at the Seventeenth Annual Meeting of the New Orleans Graduate Medical Assembly, March 10, 1954.

† From the Division of Urology in the Department of Surgery, The Medical School, Minneapolis, Minnesota.

died after catheterization did not bleed to death; nor can urethral injury explain renal failure in fatal cases. Sedillot assumed that absorption of urine was important because a dog died after an intravenous injection of urine from a prostatic; bacteriology was unknown. Albarran's "ex vacuo bleeding" ignores the flexibility of the vesical wall and the lack of suction. Desormeaux's analogy to postpartum bleeding overlooks the absence of a placenta. It is unlikely that a contracting organ can tear its own veins as suggested by Mercier.

While the idea that edema or congestion from a sudden fall of intrarenal pressure can disturb renal function allows for the frequency of uremia in fatal reactions to catheterization, it is unproved. Thus, Tandler and Zuckerkandl attribute the hydronephrosis of prostatism to an elevation of the base of the bladder which pulls the ureters up against the deferent ducts, thus compressing the former; and Duttmann and Kreutzmann believe that the hypertrophied detrusor compresses the intramural ureters. If either is correct, abrupt evacuation of the bladder cannot cause a sudden alteration of pressure within the renal pelves.

Moreover, one can produce the lesions usually attributed to sudden emptying of the bladder by ligation of the dog's urethra. Hemorrhages appear in the bladder after forty-eight and in the kidneys after seventy-two hours; these are postponed for twenty-four to forty-eight hours if the spinal cord is transected first; they are not altered by sudden decompression of the bladder. They are, therefore, due, not to emptying of the bladder, but to distention and straining. Similar changes are seen in the urinary tracts of patients dying of untreated prostatism.

Elfving has observed that vesical and renal congestion and hemorrhages as well as hematuria occur in acute urinary infections, and has attributed all of the phenomena ascribed to "sudden decompression" to acute infection. It is well known that the distended bladder affords an ideal situation for bacterial growth, especially if the prostate is traumatized with a catheter. This provides a culture medium for any bacteria already present or introduced into the urethra or prostate. It is probable, therefore, that any attempt to "decompress" the bladder will merely prolong the existing stasis and so promote progress of the infection.

Sudden deaths during emptying of the distended bladder have been blamed upon instrumentation, but without satisfactory necropsy reports excluding such complications as pulmonary embolism, coronary thrombosis, and cerebral accidents.

Many writers have ascribed untoward reactions to acute hypotension secondary to an hypothetical fall in the intrarenal pressure, but have not shown any specific causal relationship between the pressure in the renal pelves and that in the blood vessels; Young and Shaw have suggested that moderate hypotension results from the relief of discomfort through catheterization, and from the relaxation which results from deciding to have something done. Elfving believes that severe hypotension is due to acute pyelonephritis; it is probably identical with bacteremic shock.

I have studied the autopsy protocols of 71 unoperated patients dying from prostatism; I could not distinguish those whose bladders had been emptied suddenly from those subjected to gradual decompression. Eighty per cent died with renal failure secondary to combinations of acute and chronic pyelonephritis with hydronephrosis. The remaining 20 per cent died of extraurinary infections, such as pyemia, pneumonia, peritonitis, and endocarditis, but every patient in the group died of infection in some form.

Two clinical series, each of 120 patients having more than 500 cubic centimeters of residual urine, were also studied. They were treated alike except that in one group the bladder was emptied suddenly, while in the other it was evacuated gradually. The mortality of treatment (4.1 per cent, and that of subsequent prostatec-

tomy (5.3 per cent) was identical in the two series, indicating that the rate of withdrawal of the urine was of no consequence.

From the evidence enumerated, I concluded that the reactions previously attributed to sudden decompression of the chronically distended bladder resulted from infection of the damaged urinary tract quite apart from the rate of evacuation of the urine. Bazy (1896), Rose (1927), Praetorius (1929), Brecher and Chwalla (1931), Homb (1948), and Hryntschak (1949) have reached essentially similar conclusions.

The work just described was done between 1928 and 1931; since that time no distended bladder has been decompressed gradually at the University Hospital, and no ill effects attributable to the rate of emptying have been seen. It has been impossible to correlate the amount of residual urine (up to 3000 cubic centimeters) with the danger of an untoward reaction. More useful clinical guides have been the general state of the patient, his renal function, and his water, electrolyte, and protein balance.

Experience has shown that the prostatic who needs special attention presents the following picture: he has long suffered from thirst and anorexia, and has lost weight; although he is thirsty and exhibits dehydration, he is afraid to drink lest he aggravate his urinary difficulties. The bladder is distended, and the urine is pale and dilute. The hemoglobin may be normal on admission, but will fall with hydration. There is retention of nitrogen, and often of chlorides, frequently with a low plasma bicarbonate (hyperchloremic acidosis). Hypoproteinemia is fairly common, but is rarely sufficient to cause edema. Hyperpotassemia is uncommon, because anorexia reduces potassium intake as renal function fails, but the serum potassium should be measured (or an electrocardiogram secured) in the seriously ill individual to detect aberrations before they become serious.

The patient is at once given a wide spectrum antibiotic, orally if possible or parenterally if necessary. Sulfonamides and streptomycin are avoided because of the poor renal function. If a cystogram shows no diverticula in the anterior wall, a trocar cystostomy is made to provide adequate drainage without infecting the urethra and prostate. If diverticula of the anterior wall are found, an inlying catheter is inserted.

Daily weighing is instituted as a simple and fairly reliable guide to hydration and nutrition. A sudden gain of weight suggests overhydration; a steady fall without excessive loss of fluid (fever, sweating, vomiting, diarrhea, polyuria) indicates that caloric needs are not being met. Fluids are forced, parenterally if necessary, with a sharp eye on the urine output and body weight. If output keeps pace with intake, allowing for the water of the food and for the insensible fluid loss, intake is maintained at 3 to 4 liters per day until renal function stabilizes. If output remains low despite an adequate intake, an hematocrit determination is made; if this shows hemodilution and there has been an undue gain of weight, the fluid intake is reduced to the level of the output and the patient is digitalized.

Hyperchloremic acidosis will correct itself as urinary output increases. However, if it is so severe as to cause Kussmaul breathing and vomiting, one should give 5 per cent sodium bicarbonate, intravenously, according to the formula of Kagan: 55 minus the carbon dioxide combining power in volumes percent, divided by four, times the body weight in pounds, equals the number of cubic centimeters of bicarbonate needed. For milliequivalents, the corresponding formula is: $27\text{-}CO_2 \div 2$, x body weight in pounds. In seriously ill patients, it is best to give one half to two-thirds the calculated dose and then to measure the plasma bicarbonate again, for fear of overcorrecting acidosis or of producing hypernatremia.

If pronounced diuresis occurs, one watches the urinary chlorides or those of the plasma lest hypochloremic shock occur.

Normal saline is given intravenously if the patient is dehydrated, but hypertonic saline is preferred if hydration has already been attained.

The serum potassium (or electrocardiogram) is watched closely for a dangerous rise in oliguria, and for an excessive fall in severe diuresis in a patient with a poor appetite, vomiting, or diarrhea. Warning signs in each include paresthesias and weakness in the extremities. Either can cause cardiac standstill and sudden death. Oliguria calls for a potassium-free diet such as corn oil and glucose or butter balls enriched with sugar, with the object of supplying enough calories to prevent utilization of bodily protein with release of potassium. Calcium gluconate (1 gram in a 10 cubic centimeter ampoule) intravenously is of value as an antagonist to potassium; the administration of small volumes of hypertonic glucose with covering insulin may reduce the serum potassium by driving some of it into the cells, but this is of very limited value in oliguria. If oliguria persists, one must resort to some form of dialysis or to the cation resins, discussion of which is beyond the scope of this presentation.

Potassium loss in severe diuresis can be met by giving 4 to 6 grams of potassium chloride in orange or tomato juice once or twice daily; if oral administration is impossible, 2 to 5 grams of potassium chloride are added to each liter of intravenous fluid, restricting the rate of administration to 180 drops per minute to avoid dangerous levels in the blood.

A rare event in uremia from prostatism is hypocalcemia. It results from binding of calcium by phosphorus retained by the impaired kidneys, and leads to general irritability, fibrillary muscular twitchings, carpopedal spasm, and a positive Chvostek's sign. Calcium is given orally, usually as milk (but as gluconate intravenously if necessary) with large doses of vitamin D, and with 1.25 to 3.75 milligrams of dihydrotachysterol daily. Attempts are also made to improve the renal function.

While frank avitaminoses are rare in these patients, their stores are usually depleted so that it is wise to give multivitamin capsules in larger than normal doses. If oral medication is impractical, suitable preparations are added to the intravenous fluids.

Anemia is corrected by transfusions of whole blood. Hypoproteinemia demands a high protein diet. Patients unable to eat may be given protein hydrolysate intravenously, although the danger of reactions makes one hesitate in critical illness. Emergency needs may be met, in the presence of anemia, by whole blood; in its absence unpooled plasma can be used, although the fact that a depleted patient will require as much as a liter a day makes it so expensive as to contraindicate all but emergency use. In less urgent circumstances if the appetite is poor, a high protein mixture such as Varco's * may be given orally as a drip through a nasal tube. Some patients will find skimmed milk enriched with skimmed milk powder more palatable, especially if flavored with vanilla or chocolate. Diarrhea may be a problem with fluid high protein mixtures.

The objective is a man who is alert, active, bright of eye, and hungry, and who has a good output of urine in relation to intake of fluid. When he has achieved this state, he is ready for a definitive operation upon the prostate, regardless of laboratory findings. An occasional patient will not respond within a reasonable period to the regimen just outlined; failure may be due to irreparable renal damage, but a long time should be allowed for free drainage and detailed attention to fluids, electrolytes, and proteins before concluding that sufficient recovery to permit a prostatic operation will not occur. If cystostomy has not been done, it should

---

*

Whole eggs	6
Egg whites	2
Skimmed milk powder	120
Lactose or sacrose	300
Skimmed milk	1000.0 cc.
Salt	5.0 G.

One cc. contains 1.6 calories

be performed now, because advanced renal damage is more likely to improve with protracted drainage if there is no source of infection within the urethra or prostate.

With our effective antibiotics, our blood banks, and our improved but still imperfect knowledge of the importance of water, electrolytes, and nutrition, the number of neglected prostatics who cannot be salvaged sufficiently to permit a reasonably safe operation, particularly a well-executed transurethral resection, should be very small indeed.

ACUTE PANCREATITIS; CLINICAL STUDY OF ONE HUNDRED CASES *

WALTER F. BECKER, M.D.
NEW ORLEANS

Acute pancreatitis is being recognized with increasing frequency largely because of a much keener awareness of the condition and the more frequent use of the serum amylase test. The actual clinical incidence of the disease is unknown. At the Charity Hospital in New Orleans acute pancreatitis seems to be about one-tenth as common as acute mechanical intestinal obstruction and about one-fiftieth as common as acute appendicitis. This report is based upon a study of the clinical features of acute pancreatitis as observed in a series of 100 consecutive cases seen in this institution during the past decade.

MATERIAL

In the ten year period, 1943 to 1952, inclusive, there were 100 cases of acute pancreatitis seen on all services at the Charity Hospital. Only the cases of those patients are included who were proved by laparotomy or autopsy to have acute pancreatitis, or if laparotomy or autopsy was not performed, who presented a consistent clinical picture in conjunction with an increased serum amylase. Excluded from the study were 9 patients whose acute pancreatitis appeared to have been initiated by trauma to the abdominal wall or a surgical operation in the region of the pancreas, because they constitute a special group in which the pathogenesis and natural history of the disease may not be comparable to that of the usual variety of acute pancreatitis. These cases of traumatic pancreatitis have previously been reported in detail.[1]

The age distribution by decades is presented in Figure 1; the greatest incidence

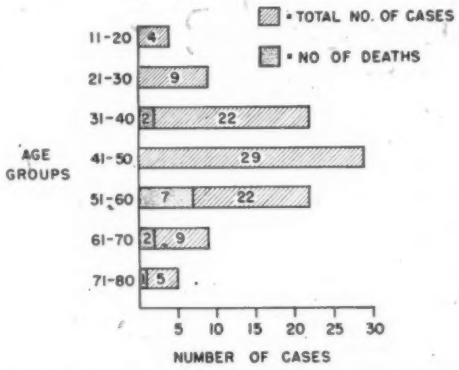

Figure 1. Age distribution by decades in 100 cases of acute pancreatitis. Patients over fifty years of age comprised 36 per cent of the material and 83 per cent of the deaths. The death rate up to and including fifty years of age was 3 per cent, above fifty years it was 27 per cent.

being in the fifth decade. The youngest patient was 16 years old; the oldest 76 years. Patients over 50 years of age comprised 36 per cent of the material and 83 per cent of the deaths.

Fifty-three per cent of the patients were males; 47 per cent were females.

Fifty-three of the patients were white; 47 were colored. This suggests a slightly increased incidence of acute pancreatitis among the whites, inasmuch as the ratio of whites to Negroes of the Charity Hospital population during the period under study was about 2 to 3.

Fifty patients had had symptoms of acute pancreatitis for more than twenty-four hours when admitted to the hospital; whereas 50 were early cases.

There were 84 cases of acute edematous pancreatitis, and 16 cases of acute pan-

* From the Department of Surgery, Louisiana State University School of Medicine, and the Charity Hospital of Louisiana at New Orleans.

creatic necrosis. Patients were considered to have acute edematous pancreatitis if at operation or autopsy there were found fat necrosis and pancreatic induration and swelling without hemorrhage or necrosis. If autopsy or operation was not performed the case was classified with the edematous group if the clinical course was benign and if such complications as pseudocyst and abscess did not occur. Thus, the 16 cases of acute pancreatic necrosis were identified at operation or autopsy.

CLINICAL MANIFESTATIONS

Despite the lack of uniformity of the clinical picture presented by patients with acute pancreatitis, the correct diagnosis can be made in the majority of cases if the surgeon is familiar with and alert to the wide variety of clinical manifestations. Paxton and Payne [4] reported from the Los Angeles County General Hospital a series of 207 cases, and on the basis of distinctive clinical patterns classified the patients into five distinct clinical groups.

In Group I were included those patients which fit the classical textbook description of acute pancreatic necrosis. Shortly after the ingestion of a heavy meal or an excessive amount of alcohol an obese middle aged person experienced sudden onset of severe steady epigastric pain with associated nausea and persistent vomiting. Cyanosis and shock were soon evident and the disease terminated fatally in a relatively short period of time. Massive myocardial infarction and perforated peptic ulcer were seriously considered in the differential diagnosis. About 10 per cent of our patients fell into this group.

Classified in Group 2 were those patients whose signs and symptoms suggested acute cholecystitis. The pain was less severe, usually became localized in the right upper quadrant, frequently radiated to the back, and was accompanied by less persistent nausea and vomiting. In many instances there was a history of previous similar but less severe attacks. About 25 per cent of our patients belonged to this group.

The cases in Group 3 presented a clinical picture resembling acute intestinal obstruction. Severe abdominal pain, nausea, vomiting, distention and obstipation were predominant features. This pattern was evident in about 20 per cent of our cases.

Placed in Group 4 were those patients with symptoms suggesting acute peptic ulceration, penetrating peptic ulcer or acute alcoholic gastritis. While epigastric pain and tenderness were present, persistent nausea and vomiting were the most striking features. Thirty per cent of the patients were in this category.

In Group 5 were the 11 patients who entered the hospital several days after the acute attack of upper abdominal pain, tenderness, nausea and vomiting, and in whom a tender epigastric or right upper quadrant mass had appeared.

Cattell and Warren [2] have suggested the addition of a sixth clinical category which would include a considerable number of patients with acute edematous pancreatitis with symptoms which are mild, ill defined and nonspecific in localization and detail. These authors suggest that it is quite possible that this type of pancreatic inflammation is the most common variety of the disease.

The fact that one third of the patients had biliary tract disease and 7 others had a peptic ulcer made it difficult to assess the past medical history relative to previous attacks of acute pancreatitis, but it was concluded with reasonable certainty that 60 per cent of the patients had previously experienced attacks of abdominal pain somewhat similar to that of the present illness.

Alcoholism has often been described as a predisposing factor in acute pancreatitis. Twenty-two per cent of the patients in this series were considered to be chronic alcoholics, and in 13 per cent the onset of acute abdominal pain was known to have followed the ingestion of alcohol.

Four patients had acute pancreatitis within three months after the termination of a normal pregnancy, and in each instance there had been repeated similar but less severe attacks during pregnancy.

Only 1 of these 4 patients had associated gallbladder disease. Langmade and Edmondson[3] recently reported 9 cases of acute pancreatitis occurring during pregnancy and the postpartum period.

The sudden onset of severe abdominal pain was the initial symptom in over 90 per cent of the cases. In 13 cases it appeared shortly after the ingestion of a heavy meal. The pain was usually severe, steady and first involved the upper abdomen. Although in 70 per cent of the cases the pain originated in the epigastrium, upon arrival at the hospital the pain had become generalized in 16 per cent of the cases. Pain was experienced only in the right upper quadrant in 3 patients, left upper quadrant in 3, right lower quadrant in 1, and left lower quadrant in none. There was radiation of pain to the back in 38 patients, right shoulder in 2, left costovertebral angle in 3 and left shoulder in 1. Diagnostic errors occurred much more commonly in those patients in whom pain was prominent in areas other than the epigastrium.

Nausea was experienced by 90 per cent of the patients, and vomiting occurred in 82 per cent. Massive hematemesis was not observed in this series, but 5 patients noticed blood-streaked vomitus.

Diarrhea was less common than constipation, being present in about 10 per cent of the cases. A history of melena was elicited in 5 cases, but this could not be confirmed by clinical or laboratory examination.

PHYSICAL SIGNS

Only about one-half (53 per cent) of the total series of patients had a temperature of 100° F. or above. A pulse rate above 90 was present in 60 per cent of the cases. In one-fifth of the patients the initial blood pressure recording was above 160/90; while the usually accepted signs of surgical shock were observed in only 8 patients. Only 3 patients were considered cyanotic at the time of admission. One-fourth of the patients were obese.

Clinical icterus was detected in only 8 patients, 6 of whom had proven biliary tract disease. Eight of the 12 patients with an elevated icterus index had associated cholecystic disease. While jaundice may result from temporary occlusion of the intrapancreatic portion of the common bile duct by pancreatic swelling and edema it may also be due to hepatic and biliary tract lesions.

The most important physical findings were referrable to the abdomen. Abdominal distention secondary to paralytic ileus was evident in a third of the patients, but this was usually a prominent feature only in the late cases and in those with severe pancreatic necrosis. In only 1 moribund patient was it impossible to elicit abdominal tenderness. In 43 of the patients the tenderness was present throughout the abdomen, in 30 it was confined to the epigastrium, only in the right upper quadrant in 5, in the right lower quadrant in 3, and only in 1 instance was tenderness confined to the left lower quadrant. Some degree of muscle spasm was present in 82 of the patients, and rebound tenderness was elicited in 90. Diminished or absent peristalsis was noted in 82 patients.

A firm, fixed, tender abdominal mass was present in 11 cases; in 6 patients it was present upon admission, and it developed later on in the course of the disease in 5 others. The mass was epigastric in location in 7 instances, and involved only the right upper quadrant in 4. The mass was in every instance a pseudocyst or abscess, and occurred only in the acute pancreatic necrosis group. One patient presented discoloration, edema and induration of the left flank (Grey Turner's sign). Discoloration of the periumbilical area (Cullen's sign) was not observed in this series of cases.

RESULTS

There were 12 deaths in the series of 100 cases, a mortality rate of 12 per cent. Three patients were moribund on admission but are included in these totals. This mortality compares favorably with that reported from most large general hospitals (Table 1).

The major factors influencing prognos-

TABLE 1
MORTALITY OF ACUTE PANCREATITIS

INSTITUTION	YEARS	CASES	MORT. RATE
Mass. General Hospital [5]	1946-51	134	12%
Cincinnati Gen. Hospital [6]	1941-50	164	14%
Los Angeles Co. Gen. Hospital	1933-46	307	33.3%
Charity Hospital (Present Series)	1943-52	100	12%

is were the age and race of the patient and the clinico-pathological type of the pancreatitis.

Patients over 50 years of age comprise 36 per cent of the material and 83 per cent of the deaths. The death rate up to and including 50 years of age was 3.1 per cent; above 50 years it was almost ten times as high (27.7 per cent).

The mortality rate among the white patients was almost twice as high as it was in the Negro group—15.1 per cent compared with 8.5 per cent.

Of the 84 patients given a diagnosis of acute edematous pancreatitis only 3.6 per cent died. The mortality was 56.3 per cent in the group with pancreatic necrosis.

The mortality rate for the group of 34 patients treated by early operation was 14.3 per cent, and for the 66 patients managed conservatively it was 10.7 per cent.

Nine of the 16 patients with acute pancreatic necrosis had early operation with a mortality rate of 33.3 per cent; but the mortality rate was 85 per cent in the small group of patients with pancreatic necrosis treated conservatively.

Almost 70 per cent of the cases of acute edematous pancreatitis were managed conservatively with a mortality rate of only 1.7 per cent; while operation in this group was attended by a mortality rate of 7.7 per cent.

These data concerning the influence of operation upon mortality are really of no statistical value because of the small number of cases involved and the fact that in the absence of operative or autopsy verification patients were arbitrarily placed in the acute edematous pancreatitis group if the clinical course was benign and if there was no evidence of hemorrhage, necrosis, or suppuration in or about the pancreas.

Major complications were observed in almost one-half of these 100 cases of acute pancreatitis. A pseudocyst developed in 5 cases, abscess in or about the pancreas in 5, external pancreatic fistula in 4, pancreatolithiasis in 3, and hydrothorax in 2. At least 19 patients have continued to have recurrent attacks of abdominal pain of sufficient severity and specificity to strongly suggest the diagnosis of chronic relapsing pancreatitis. Transient or permanent diabetes was observed in several patients.

DISCUSSION

During the years covered by this study conservative or nonoperative treatment has been considered the preferred method of management of patients with acute pancreatitis. Early operation has been favored in those instances in which the diagnosis could not be established with reasonable certainty or when there existed signs of obstruction of the biliary tract, suppuration, and cystic accumulations. It thus becomes necessary to explain how it happens that one-third of the patients with acute pancreatitis were treated by early operation during a period when the conservative management of this condition was considered at this hospital to be superior. Errors in preoperative diagnosis seem to have accounted for three-fourths of these operations. This poor diagnostic showing can in part be ascribed to the fact that during the early years of this study facilities for the performance of the serum amylase test were not available in this institution at night. A preoperative determination of the serum amylase was performed in only 18 of the 34 patients subjected to operation. A few of the operations were performed because of the opinion that early decompression of the biliary tract would favorably influence the course of the disease. Cholecystectomy plus choledochostomy was

carried out in 4 patients; cholecystostomy was done in 8 instances; and choledochostomy alone was employed in 1 case.

An analysis of these 100 cases of acute pancreatitis does not provide significant statistical data to assist in the resolution of the controversy of operative versus nonoperative treatment of the disease. It is interesting that almost one-half (16) of the 34 patients subjected to operation had only an exploratory laparotomy, a procedure which is not likely to have favorably influenced the condition of these seriously ill patients.

SUMMARY AND CONCLUSION

A clinical review of 100 consecutive cases of acute pancreatitis observed at Charity Hospital during the past decade has been presented.

The mortality for the entire series was 12 per cent; for the 84 cases of acute edematous pancreatitis it was 3.6 per cent, and for the 16 cases of acute pancreatic necrosis it was 56.3 per cent.

The diversity of the clinical and pathological manifestations of the disease has been emphasized.

One-third of the patients were subjected to early operation, in most instances because of errors in dignosis.

The treatment of choice in most cases of acute pancreatitis is conservative. Early operation should perhaps be restricted to those instances in which the diagnosis cannot be established with reasonable certainty or when there exists signs of biliary tract obstruction, suppuration or cystic accumulations.

REFERENCES

1. Becker, Walter F.: Traumatic Pancreatitis. Amer. Surgeon (In Press).

2. Cattell, R. B., and Warren, K. W.: Surgery of the Pancreas, W. B. Saunders Co. 1953.

3. Langmade, C. F., and Edmondson, H. A.: Acute pancreatitis during pregnancy and the postpartum period. Surg., Gynec. and Obst. 82: 43 1951.

4. Paxton, J. R., and Payne, J. H.: Acute pancreatitis. A statistical review of 307 established cases of acute pancreatitis, Surg., Gynec. and Obst. 86:69, 1948.

5. Raker, J. W.; and Bartlett, M. K.: Acute pancreatitis, New England J. Med. 249:751, (Nov. 5) 1953.

6. Silver, V. E., and Wulsin, J. H.: Consideration of the lethal factors in acute pancreatitis. Arch. of Surg. 63:496 1951.

CURRENT STATUS OF THE SURGERY OF MITRAL STENOSIS WITH A REPORT OF TWENTY-SEVEN CASES *

H. REICHARD KAHLE, M. D.,
PAUL T. DeCAMP, M. D.
NEW ORLEANS

The progress made in cardiovascular surgery over the past few years has removed it from the realm of the rare and the spectacular and has brought it to a position comparable with that of other types of major surgery. Responsibility for determining whether or not operation shall be recommended in the cardiac conditions amenable to surgery rests squarely upon the shoulders of every physician who treats heart disease. The surgical mortality of cardiac surgery is now acceptably low. To advise against operation in the face of that fact is culpable and unjustified. The risk of the patient who must live with his disease is frequently much greater than the risk he would undergo if he submitted to surgical correction for it.

Any discussion of the cardiac diseases amenable to surgery properly begins with mitral stenosis, which is the most frequent condition in that category. The purpose of this paper is to review the current status of surgery for it, and to present a brief summary of our own experiences.

White [1] is responsible for the estimate that from 0.5 to 1 per cent of the population of the northeastern United States and northern Europe suffers from valvular heart disease, and that perhaps 85 per cent of all persons affected with rheumatic heart disease develop some degree of valvular deformity, most often in the mitral valve. Congenital cardiac lesions constitute only 2 per cent of all instances of heart disease. Rheumatic fever, on the other hand, causes 25 per cent of all cardiac conditions observed in hospitals, and in half of them mitral stenosis is the predominant lesion.

* From the Department of Surgery, Tulane University of Louisiana School of Medicine, and the Ochsner Clinic, New Orleans.

These figures have practical implications. Because congenital heart lesions are so infrequent, it is best that their operative relief be undertaken only in a few special centers. It would, however, be completely impractical for all patients who require mitral commissurotomy to be treated in a limited number of centers; there are too many of them. As a result, the operation has become a standard procedure, which can be carried out in all thoracic surgical clinics in large medical centers. One should not overlook the significance of a recent report on cardiac surgery "in a community general hospital." [2]

HISTORICAL NOTE

It is undoubtedly because of the frequency of mitral stenosis that the earliest efforts to correct a heart lesion by surgical measures were directed to the mitral valve. [3, 4] In 1898, Samways made the suggestion that surgeons would soon be operating on it. In 1902, after working on the problem experimentally, Sir Lauder Brunton made the concrete suggestion that mitral stenosis be corrected surgically. He stirred up a fine commotion, which continued for a period of weeks, in the correspondence columns of the *Lancet*. During the next two decades additional experiments were conducted at Peter Bent Brigham Hospital in Boston, Harvey Cushing being among those who worked on the problem, but it was not until 1924 that Cutler, Levine and Beck actually operated for mitral stenosis. The attempt was short-lived, for the compelling reason that they had only 1 survival in 7 operations.

In the light of present knowledge it is easy to understand why these pioneering surgeons failed. For one thing, their approach was through the ventricle; whereas one of the cardinal principles which has been learned by cardiac surgeons through hard experience is that conical valvular lesions are better approached from the base than from the apical side. For another, the early surgeons did not appreciate the necessity for avoiding mitral regurgitation. When Smithy, in 1950, again attacked the valve, he failed for precisely the same reasons. In 1925, however, Souttar anticipated the modern approach, through the left auricular appendage, and his patient not only survived dilatation of the stenotic mitral ring but was greatly improved afterward.

It is sad to think of the years which were wasted between Souttar's succesful case and the revival of interest in this field in the last four or five years. His report attracted almost no attention, probably because it concerned only a single case, and nothing more was done to solve the problem of mitral stenosis by surgical measures until Bailey, Glover and their associates in Philadelphia [5] and Harken and his co-workers, [6] again at Peter Bent Brigham Hospital in Boston, demonstrated, beyond argument, that acquired valvular disease is amenable to surgery and also showed, which is just as important, that the operation is actually attended with less risk than many other procedures that are today accepted as commonplaces of surgery.

ANATOMY, PHYSIOLOGY AND PATHOLOGIC PHYSIOLOGY

The mitral valve is a bicuspid valve made up of a larger anteromedial leaflet and a smaller posterolateral leaflet. By experimental damage of these leaflets Harken and his group [7] and Hufnagel and Gillespie [8] were able to show, independently, that the anteromedial leaflet is the more important. Anatomically, this is what would be expected, since it forms approximately two-thirds of the floor of the left auricle. The observations of these two groups also bear out what has been learned of the normal functioning of the valve from in vivo studies, in contrast to earlier ideas derived from the study of the fixed heart after death: During diastole the anterior leaflet opens and its base swings medially, partly occluding the aortic outlet tract and opening the floor of the auricle, just as if a trap door had been sprung. In systole, the blood is deflected from the under surface of this leaflet into the aorta, the anterior cusp

acting, as Harken expresses it, as a baffle.

Once the mitral valve becomes fused in rheumatic heart disease, the free motion just described is restricted and the valve cannot open beyond a certain point. When, therefore, an increased flow of blood is required in response to an increased demand, it can be obtained in only one of two ways: A longer time must be allowed for filling, or the pressure in the left auricle must increase, so that the blood will be forced through faster. Both of these mechanisms are operative in mitral stenosis. When stenosis is present, pressure in the left auricle necessarily increases and, also necessarily, since the pulmonary veins contain no valves, it is transmitted back to the pulmonary capillaries. Two other mechanisms then come into play. The first is a decrease in the cardiac output. The second is pulmonary hypertension, which does not usually occur until the area of the mitral valve has been reduced from a normal of 6 sq. cm. to about 1 sq. cm., which is approximately 20 per cent of normal. Pulmonary hypertension is accompanied by narrowing of the smaller pulmonary arteries and arterioles, which undergo pathologic changes, chiefly in the form of intimal proliferation and medial thickening. As a result of these changes, there is increased resistance to the blood flow through the affected segment of the lung, and the next development is a work hypertrophy of the right ventricle.

Patients with so-called tight mitral stenosis vary considerably in the manifestation of pulmonary resistance. Increased arteriolar resistance tends to protect against the development of pulmonary edema, but its appearance is accompanied by certain disadvantages. Patients who present it are likely to show signs of a decreased cardiac output, as manifested by fatigue, exhaustion, weakness and listlessness. In the final stages of their disease, because of decompensation of the overworked right ventricle, they are also more prone to congestive failure. According to Dexter,[9] when pulmonary resistance has increased to five or ten times normal, cardiac enlargement occurs. When it has increased to ten or fifteen times normal, gross right ventricular failure is likely.

Patients who do not develop increased pulmonary resistance are prone to bouts of pulmonary edema whenever pressure in the pulmonary capillaries rises above that exerted by the osmotic effect of the plasma proteins (Starling's law). They show little or no evidence of cardiac enlargement but they present the symptoms typically associated with mitral stenosis, namely, dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and hemoptysis.

As pulmonary resistance increases and the burden is shifted from the pulmonary capillaries to the right ventricle, patients who formerly presented pulmonary edema and its associated symptoms and signs begin to show improvement from that standpoint, but they present, instead, symptoms and signs of lowered cardiac output. Often the balance between the two mechanisms is extremely delicate.

Rheumatic valvular disease causes a thickening of the free edges of the mitral valve. Somewhat later, these edges become fused. Gradually, therefore, what was once a flexible slit becomes a more or less fixed and rigid ring of gradually narrowing diameter. The chordae tendinae may become thickened and shortened. The valve edges, in addition to becoming thickened and fused, may also become nodular and calcified. In most instances, the pathologic process is not complete. The periphery of the valve, close to its attachment to the auricular-ventricular ring, usually remains flexible. For this reason, restoration of valvular function can be expected after the commissures have been freed at operation.

Gorlin and Gorlin [10] have devised mathematical formulas by which the area of the valvular orifice can be calculated in the living subject. They are based upon the physical laws governing flow through fixed orifices and are dependent upon flow rate, the pressure gradient across the orifice, and certain known coefficients.

The limitation of activity suffered by patients with mitral stenosis can be correlated with the decrease of the valvular cross sectional areas but most cardiac surgeons, less scientifically, are content to estimate that when the patient has symptoms sufficiently severe to justify a resort to surgery, the mitral opening is from 0.5 to 1 cm. in diameter and barely admits the tip of the index finger.

DIAGNOSIS AND SELECTION OF CASES

The signs and symptoms of mitral stenosis are so well known that it is scarcely necessary to enlarge upon them. The most typical physical signs are a rumbling, crescendo presystolic murmur at the apex and a diastolic murmur (Graham-Steel murmur) at the pulmonic area. The diastolic murmur is presumably the result of functional pulmonary insufficiency secondary to dilatation of the pulmonary artery.

In pure (uncomplicated) mitral stenosis, cardiac enlargement is usually limited to some dilatation of the left auricle and, in advanced cases, hypertrophy of the right ventricle. If enlargement of the left ventricle occurs, mitral regurgitation, or some other type of valvular disease, should be suspected. Auricular fibrillation is common.

Electrocardiography may show changes in the P wave, as the result of left auricular dilatation, and right axis deviation may also be present. Cardiac catheterization is useful in determining the degree of pulmonary hypertension present, but it is not an essential part of the preoperative investigation. It is well, however, to perform it whenever possible at this time; considerably more data than now exist must be collected about the results of surgery for mitral stenosis, and this method permits an objective study of improvement not possible by any other test. As Baker and his associates have warned,[11] it is not entirely free from risk.

The best method of diagnosis, as Bourne[12] has stressed, is to take a really full history and to ask enough questions, in enough detail, to find out exactly what the patient can tolerate in the way of physical activity and exactly how much pulmonary congestion exercise causes. The only condition of differential diagnostic importance is mitral incompetence. The distinction is sometimes difficult, as our own experience proves, but it is essential, since at the present time there is no effective surgical treatment for the latter condition.

The present consensus is that mitral commissurotomy should be performed in patients who are disabled by their disease and whose disability tends to be progressive. Patients with mitral stenosis who are asymptomatic do not need surgery. Baker and his associates[11] state that the various manifestations of pulmonary congestion provide the commonest indications for operation, as well as the most clearcut. They grade their patients from 0, with no disability, to 4, with complete incapacity, and feel that even patients in class 4 will withstand surgery well provided that the intracardiac obstruction is relieved. According to Dexter,[9] the fundamental indication for the operation is the appearance of symptoms which occur when the mitral valvular area is reduced to 1 sq. cm. or less (tight mitral stenosis).

Most cardiac surgeons prefer to operate for mitral stenosis between the ages of 20 and 40 years, but some, such as Gerbode and his associates[4] at Stanford University, report that some of their most dramatic results have occurred in patients up to 55 years of age.

There was formerly general agreement that certain associated diseases and conditions furnished absolute contraindications to surgery for mitral stenosis. These included active rheumatic fever, active subacute bacterial endocarditis, severe aortic valvular disease (regurgitation or stenosis), and severe mitral regurgitation.

There is, however, a notable tendency in the recent literature toward increasing flexibility in the acceptance of patients for operation. Wilcox and Grace,[13] writing in 1951, stated that they would "unhesitatingly" drop from consideration patients with mitral regurgitation or evi-

dence of calcium deposition in the mitral valve. Baker and his associates,[11] writing in 1952, reported that some degree of calcium was present in 40 of their 100 cases. Results were admittedly not as good in this group, but they were poor only when calcification was really extensive. These observers felt that this pathologic change was responsible for only 3 of the 9 unsatisfactory results in their first 50 cases. They are equally liberal in respect to mitral regurgitation; their feeling is that a small amount is necessarily present whenever the orifice is small and rigid but that commissurotomy may restore almost normal functioning in closing as well as opening of the valve. Bailey and his group[14] have performed a number (28) of successful operations for mitral stenosis combined with severe mitral regurgitation, either at the same sitting or during the same period of hospitalization. They are also operating under the same circumstances for mitral stenosis combined with aortic stenosis.

Auricular fibrillation is not in itself a contraindication to surgery, though, since it occurs only in advanced disease, the prognosis is correspondingly less hopeful when it is present. It was observed in 44 of the 100 cases reported by Baker and his associates,[11] and embolism was responsible for 4 of the 8 deaths in this group.

Congestive failure is also not a contraindication to surgery in mitral stenosis provided that it has not been present too long and that it responds to medical measures. It was a complication in 13 of the first 50 cases operated on by Baker and his associates,[11] and was associated with auricular fibrillation in all but one of these.

A history of embolism is another observation which is no longer regarded as a contraindication to operation. As Dexter[9] notes, a higher incidence of this complication can be expected in patients who are fibrillating, but the risk is worthwhile, for these are the patients who are likely to receive the greatest benefit from surgery. Baker and his associates[11] take much the same position. As their series shows, the history is not always reliable: A thrombus was found at operation in only 9 of the 17 cases in which a story of embolism had been secured. Embolism was a postoperative complication, however, in 7 of their 100 cases, and was responsible for 4 deaths.

Bourne[12] makes the sound suggestion that there be careful assessment of the myocardial status before surgery is undertaken. As he points out, the myocardial degeneration produced by rheumatic disease varies enormously from one case to another and may be roughly estimated from the size of the heart. When the heart is barely enlarged, whatever symptoms the patient complains of may be attributed almost entirely to mitral stenosis. When it is greatly enlarged, the reverse is true and the symptoms are more likely to be the result of myocardial disease than of the valvular lesion. Commissurotomy would be of little if any benefit in the latter type of case.

SURGICAL TECHNIQUE

The approach to the mitral lesion is usually through the left fourth or fifth intercostal space. When the pericardial sac is opened, a pursestring of heavy silk is applied at the base of the left auricular appendage. Then a noncrushing clamp is applied and the tip of the appendage is excised, an opening being left just large enough to admit the index finger. As the clamp is released, the right index finger is slipped through the opening down to the valve and the precise conditions present are determined by palpation. The finger may be kept in the auricular chamber almost indefinitely without ill effects, but the stenotic opening of the valve, for obvious reasons, must not be occluded for more than a few seconds at a time.

There are two methods for opening the commissures of the valve, one instrumental and the other digital. Harken[7] has popularized what he terms finger fracture valvulotomy, which is dependent for success upon the possibility of opening the

commissure by digital manipulation along the original line of adherence. While this is the more popular of the two methods, it is not always possible. Not infrequently it will be found that finger fracture valvulotomy cannot be carried to success without taking an undue risk of tearing into one or the other of the mitral cusps because the tough, resilient ring resists all efforts to start cleavage along the original lines. Bailey, Glover and others of the Philadelphia school [14] frequently use a special guillotine type of knife which is maintained along the anterior surface of the index finger by means of a second glove worn over the first. They used this technique in 324 of the 501 cases included in their most recent report.

MORTALITY

It is unfair to discuss the surgical mortality of mitral stenosis unless there is a sharp differentiation between the early cases, in which the surgeons were literally pioneering, and the later cases, in which they built upon the foundations which these pioneers had laid. Thus Fell and his associates [3] lost 2 of their first 10 patients but had no deaths in the next 18 operations. Baker and his group [11] lost 7 of their first 20 patients but had only 6 deaths in the next 80 operations. Gerbode and his associates [4] lost 5 of their 44 patients, but 2 of these deaths taught them the extreme importance of liberalizing a severely restricted salt intake just before operation.

The safety of surgery for mitral stenosis was strikingly evident at the 1952 European Congress of Cardiology, [15] when Sellors reported 64 operations with no deaths and O'Neill reported a mortality of only 4 per cent in the 800 mitral commissurotomies performed by himself, Bailey, Glover and others of the Philadelphia group.

ANALYSIS OF POSTOPERATIVE RESULTS

While clinical improvement is the objective in all surgery for mitral stenosis, the results of operation cannot be estimated on clinical improvement alone. There are a number of other questions which must be asked, though some of them, such as whether or not the secondary alterations in the pulmonary vessels are reversible, can be answered only indirectly at the present time.

In an endeavor to secure the answer to these various questions, Dexter [9] made a careful study of the pathological physiology before and after operation in 12 patients submitted to surgery for mitral stenosis at Peter Bent Brigham Hospital. He found that in each instance the size of the mitral orifice was significantly increased. Mitral regurgitation was increased in only 1 case, in which, because of a technical error, the aortic leaflet was damaged. There was little increase in cardiac output at first, but as time went on there seemed to be a progressive increase. The same observation was also made by Baker, Brock, Campbell and Wood. [1] Dexter noted a dramatic fall in pulmonary capillary pressure in all cases but the one, just mentioned, in which mitral regurgitation was produced surgically. His studies showed that reduction in pulmonary arteriolar resistance and improvement in right ventricular function were not always striking, at least over the period of observation. Whether there will be improvement in these respects after a longer time has passed remains to be seen. Gerbode and his associates [4] catheterized 12 patients after operation and found an increase in cardiac output both at rest and with exercise. They also observed that the pulmonary arterial pressure was 20 to 40 per cent lower than before operation.

One point that should be emphasized is that clinical improvement, substantiated by such evidences of physiologic improvement as have been cited, is not necessarily paralleled by improvement in the physical findings. Cardiac murmurs, in particular, may continue entirely unchanged.

It is right and proper that clinical improvement should be correlated with physiologic studies, if only because subjective proof is always suspect when it is unsupported. This does not mean, however, that

clinical improvement is not the end and aim of all surgery for mitral stenosis. All of us working in this field know that it is. None have expressed it with such warm humanity as Baker and his associates [11] in a recent communication in the *British Medical Journal*. The need for operative relief in this condition, they said, had been clearly borne out by the number of patients who presented themselves for consideration as soon as their first report appeared. They came themselves and they were referred by general practitioners and cardiologists. The future that previously had seemed so bleak for these men and women, many of them actually or relatively young, was now completely changed. The individual tragedies of mitral stenosis are not fully realized until patients with the disease are dealt with in large numbers. A laborer, for instance, had been out of work for a year because of recurrent congestive cardiac failure. He had not missed a day from work in the five months that had elapsed since operation. A patient who before operation could not walk the length of the ward during convalescence missed his bus, after running 30 yards for it, then walked home, a distance of 4 miles, and had no trouble at all. One woman had a successful pregnancy after operation. Another was operated on successfully in the fifth month of her pregnancy. "It is doubtful," Baker and his associates conclude, "if we, as doctors, have appreciated how much his disability means to the patient. To be unable to hurry or climb stairs without paying a penalty, to forego healthy physical pursuits, to have to sleep propped up in order to avoid suffocating at night, and to live in constant fear of a stroke, of hemorrhage from the lungs, of drowning in his own expectoration, or of becoming water-logged, are just a few of his troubles. What a load is here for anyone to carry and what a nightmare for the apprehensive. No wonder that patients seize the chance that surgery now offers."

ANALYSIS OF CASES

Since March, 1951, we have performed 27 operations for mitral stenosis (Table 1) on 23 women and 4 men, who ranged in age from 17 to 50 years. The notably higher number of women is a reflection of the greater incidence of rheumatic fever in this sex. Twenty-three patients were white and 4 colored, but since 15 operations were done at Foundation Hospital, which admits only white patients, the figures are no indication of the racial incidence of mitral stenosis.

Every patient in the series either presented signs of cardiac failure on admission or had a story of episodes of cardiac failure in the past. In 19, a clearcut history was elicited of the bouts of pulmonary edema characteristic of mitral stenosis. One patient had an episode of this kind with each menstrual period. Twelve patients had a history of hemoptysis. As these stories indicate, our policy is to accept for surgery only the patients who have progressive symptoms referable to mitral stenosis; operation is never undertaken in patients who present the lesion but who have no symptoms and signs referable to it.

Fibrillation was not regarded as a contraindication to surgery. It was recognized that cardiac efficiency is decreased when it is present, and that there is a risk, when operation is undertaken, of thrombosis of the auricular appendage, and therefore, of peripheral embolism. This happened in the most recent case in our series, in which the procedure was carried out with unusual smoothness. The patient presented auricular fibrillation both before and after operation. Hemiplegia was noted the morning after operation, and death occurred within 36 hours. At autopsy a small thrombotic area at the base of the left auricular appendage was thought to be the origin of the massive, lethal embolus to the left middle cerebral artery.

Fibrillation was present in a total of 9 cases before operation and was noted in 13—not always the same cases—during some phase of the postoperative period. In 4 of these there was a reversion to nor-

TABLE 1

ESSENTIAL DATA IN 27 SURGICAL CASES OF MITRAL STENOSIS

Case No. *	Race, Sex Age	Pulmonary Edema	Hemoptysis	Fibrillation Before	Fibrillation After	Stenotic Orifice (in Mm.) Before	Stenotic Orifice (in Mm.) After	Regurgitation Before	Regurgitation After	Duration of Follow-Up and Results to Date * *	Last Observation
1	WM, 18				X	Regurgitation only		XXXX	XXXX	7 months. Unimproved.	October 1952
2 *	WF, 30			X		?	30 x 20			27 months. XXXX improvement.	July 1953
3	WM, 34			X		Valve calcified				Death 17th postoperative day, cardiac failure.	
4 *	WF, 39	X			X	?	25 x 25			17 months. XXXX improvement	July 1953
5	WF, 38	X	X			4 x 2	25 x 20			14 months. XXX improvement	July 1953
6	WF, 43			X	X	8 x 3	25 x 15			6 months. XX improvement.	October 1952
7 *	WF, 30	X	X			9 x 1	25 x 25		X	14 months. XXXX improvement.	July 1953
8 *	CF, 30	X				10 x 8	55 x 30			12 months. XXXX improvement.	July 1953
9 *	WF, 37	X	X		X	10 x 8	20 x 20	X	X	9 months. XXXX improvement.	July 1953
10	WF, 36			X		10 x 4	25 x 15	XX	XX	4 months. XXX improvement.	April 1953
11 *	CF, 35	X	X		†	10 x 8	20 x 20			7 months. XXXX improvement.	July 1953
12	WF, 30	X	X		†	6 x 3	15 x 10			5½ months. XXX improvement.	June 1953
13	WF, 50	X		X	X	12 x 4	20 x 15			4 months. XX improvement.	May 1953
14	WF, 30	X	X			12 x 5	25 x 20			2½ months. XX improvement.	April 1953
15	WF, 44	X			†	10 x 14	30 x 20			2½ months. XXX improvement.	April 1953
16	WF, 38			X		9 x 4	19 x 14	X	XX	3 months. XXX improvement.	May 1953
17 *	CF, 30	X				9 x 4	20 x 15			Death 11th postoperative day, septicemia, thrombosis of valve.	
18 *	CF, 30	X	X			9 x 4	20 x 20	X	X	2 months. XX improvement.	May 1953
19	WM, 38	X	X			13 x 4	28 x 18	X	XX	2½ months. XX improvement.	June 1953
20	WF, 36	X	X		†	8 x 3	23 x 17			4 months. XXX improvement.	July 1953
21 *	WM, 40	X	X	X	X	12 x 4	30 x 20			3 months. XXXX improvement.	July 1953
22	WF, 43					8 x 3	19 x 14	X	XX	No follow-up. In good condition on discharge.	
23	WF, 42	X		X	X	20 x 15 Regurgitation only	20 x 15	XXX	XXX	No follow-up. Unimproved when last heard from.	
24	WF, 44	X			X	8 x 3	35 x 25		XX	3 months. XXX improvement	July 1953
25 *	WF, 17	X	X			3 x 6	18 x 14			1 month. XXX improvement.	July 1953
26 *	WF, 37	X	X			10 x 4	20 x 20			3 weeks. Too early for evaluation.	
27 *	WF, 34	X ***	X	X	X	6 x 4	30 x 20		XX	Died 48 hours postoperative, embolus from auricle to left middle cerebral artery.	

* Charity Hospital.

** Graded in degree of excellence from X to XXXX.

*** At period only.

† Transient fibrillation postoperatively

mal rhythm either spontaneously or after quinidine administration. Fibrillation is a disturbing complication, no matter when it occurs, but it does not mitigate against a good result if the valve has been adequately opened. The resulting decrease in cardiac output is more than offset by the increase which results from relief of the stenosis.

Two cases in this series illustrate that the distinction between pure stenosis and mitral regurgitation is not always easy to make when a patient is being evaluated for surgery. One case (No. 1) was found, at operation, to present more insufficiency than stenosis. A finger was put through the mitral ring, but the feeling that little had been accomplished was borne out by the subsequent clinical course. The patient was alive when he was last heard from, seven months after operation, but he was not doing well. In the second case in which no significant amount of stenosis was found at operation (No. 23), the orifice was estimated to measure 20 by 15 mm. No attempt at dilatation was made. The patient was discharged in good condition fifteen days after operation, but no improvement in his condition is expected. In this case the presence of a systolic mitral murmur made it uncertain whether his difficulties were due to stenosis more than to insufficiency, but operation was undertaken because it was felt that he might be benefited by relief of the stenosis even though some regurgitation was also present.

Except in these 2 cases, and in another case (No. 3) in which a great deal of calcification was present, it was possible in every instance to produce a striking increase in the size of the stenotic ring at operation. Almost universally the orifice admitted only the tip of the index finger at the beginning of the operation, thus bearing out the general opinion that patients will show severe disability when the area of the opening is less than 1 sq. cm. When the ring is increased in size to 25 by 20 mm., it is estimated that the valve area is increased to better than 3 sq. cm., which is adequate to take care of the ordinary demands of life, including moderate exercise. These estimates are based on the principle that the area increases as the square of the radius if the opening is assumed to be circular. They can be made accurately only if there is precise knowledge of the diameter of the surgeon's index finger at various levels.

Case No. 3 illustrates how calcification and rigidity of the orifice can totally defeat the objectives of the operation. In this instance the valve leaflets as well as the stenotic ring were thickened, rigid, and markedly calcified. Although the commissures were split, it was not felt that much had been accomplished. The patient died at home, apparently of cardiac failure, seventeen days after operation, and postmortem observations confirmed the operative findings.

Regurgitation is fairly frequent but is not necessarily significant. Minor degrees are well tolerated. In the 2 cases already mentioned (Nos. 1 and 23), in which it was found to be severe at exploration, no real attempt was made to dilate the ring. In 3 other cases (Nos. 9, 10, and 18) a slight to moderate regurgitant jet was present but was not increased by surgery, and excellent clinical improvement occurred in all.

Three other patients who had no palpable regurgitant jet before the ring was opened showed some evidence of it after operation, estimated as 1 plus in one instance and as 2 plus in the other 2 cases. One patient (No. 27) died of cerebral embolism, and the clinical effect of the jet could not be evaluated. The other 2 (Nos. 7 and 24) have shown satisfactory improvement, which experience in other cases suggests is likely to continue. Three patients who presented a 1 plus jet before operation presented a 2 plus jet after operation. In 1 case (No. 22) there is no follow-up. In the other 2 (Nos. 16 and 19) clinical improvement has been progressive.

Two patients died in the hospital. One death (No. 17) occurred in a 30 year old

negro woman on the eleventh postoperative day. Her fever had been persistently high after operation and Enterococcus was reported on blood culture. At postmortem a thrombus was found on the fractured valve surface. Since this experience antibiotic therapy has been employed intensively for forty-eight hours before operation and for a minimum of five or six days afterward, even if no indication for it exists. The second death (No. 27), from an embolus of the left middle cerebral artery, has already been described. The valve ring was widely opened in this case and excellent clinical improvement would undoubtedly have occurred had the patient lived. The third death (No. 3), which occurred at home, on the seventeenth postoperative day, from cardiac failure, has also been described. The dense calcification present in this case would have prevented any benefit from surgery, although the commissures were opened, had the patient lived.

In the 2 cases, (Nos. 1 and 23) in which there was more regurgitation than stenosis, no relief was expected and none was accomplished, as has already been pointed out. One patient (No. 26) has been operated on too recently for an evaluation of her condition, though she feels better. Another patient (No. 25) was operated on only a month ago, but from a preoperative exercise tolerance of only one block, she is now able to walk the length of the Midway at Pontchartrain Beach (about four blocks) without distress. This performance, incidentally, was strictly against instructions, it being our policy to insist on restricted activity for at least six months after operation, to give the myocardium a chance for stabilization. In one other case, in which operation was done four months ago, the patient could not be located for evaluation of her current status, but the degree of valvular opening accomplished suggests that the result is likely to be good.

In the other 19 patients postoperative improvement has varied from extremely satisfactory to actually dramatic. One patient (No. 7), for instance, was a cardiac cripple when she was first seen. Her condition had become progressively worse over a three-year period. She had a severe hemoptysis on the way to the hospital and was dyspneic, cyanotic, and in failure when she was admitted. When she was examined in July 1953, fourteen months after mitral commissurotomy, she reported that she no longer suffered from edema, shortness of breath, hemoptysis, or any other symptom, that she was doing her own housework, and that even dancing produced no difficulties.

Dramatic results of this kind did not occur in all of the 19 patients who showed improvement after operation, but most of them have found a new way of living and some hope in living. Furthermore, since maximum improvement usually requires from six months to a year to become evident, and since improvement, frequently of considerable degree, has been observed in 12 (of the 16) patients operated on since January 1953, that is, during the past seven months, there is ground for optimism and for hope that as time passes, the results in these cases will be even better than they now seem to be.

SUMMARY

Mitral stenosis is the most frequent of all cardiac conditions which are amenable to surgery. Mitral commissurotomy is now associated with so low a mortality that the risk of operation is less than the risk of permitting the patient to continue to live with his disability. Any physician who treats cardiac disease must therefore assume the responsibility of advising surgery, or recommending against it, in all instances of mitral stenosis.

The essential surgical aspects of the condition are briefly outlined, with special emphasis on the proper selection of patients for operation, and a report is made on 27 personally managed cases.

Note: Since this article was submitted for publication, six additional operations have been done, with no deaths, and with immediate good results in all cases.

REFERENCES

1. White, Paul D.: Chronic valvular cardiac disease, In Cecil, Russell L.: A Textbook of Medicine, ed. 7. W. B. Saunders Company, Philadelphia and London, 1947.
2. Antopius, Nicholas A., Murray, Harrold A., O'Connor, Bernard A., and Crecca, Anthony D.: Cardiac surgery in a community general hospital, J. A. M. A. 151:8, 1953.
3. Fell, Egbert H., Paul, Oglesby; Campbell, James A., Davis, Carl B., Jr., Selverstone, Louis, and Grisson, Robert: Mitral stenosis. Physiological studies, diagnosis and treatment, Arch. Surg. 65:128, 1952.
4. Gerbode, Frank; Holman, Emile, and Hultgen, Herbert: Treatment of mitral stenosis by finger fracture valvulotomy, Arch. Surg. 65:113, 1952.
5. Bailey, Charles P.: The surgical treatment of mitral stenosis (mitral commissurotomy). Dis. Chest 15:377, 1949.
6. Harken, Dwight E., Ellis, Laurence B., Ware, Paul F., and Norman, Leona R.: Surgical treatment of mitral stenosis. I. Valvuloplasty, New England J. Med. 239:801, 1948.
7. Harken, Dwight E., Ellis, Laurence B., and Norman, Leona R.: The surgical treatment of mitral stenosis. II. Progress in developing a controlled valvuloplastic technique, J. Thorac. Surg. 19:1, 1950.
8. Hufnagel, Charles A., and Gillespie, John F.: Principles of surgical treatment in mitral stenosis, Bull. Georgetown Univ. M. Center 4:131, 1951.
9. Dexter, Lewis: Pathologic physiology of mitral stenosis and its surgical implications, Bull. New York Acad. Med. 28:90, 1952.
10. Gorlin, R., and Gorlin, S. G.: Hydraulic formula for calculation of the area of the stenotic mitral valve, other cardiac valves, and central circulatory shunts, Am. Heart J. 41:1, 1951.
11. Baker, Charles, Brock, R. C., Campbell, Maurice, and Wood, Paul: Valvotomy for mitral stenosis. A further report on 100 cases, Brit. M. J. 1:1043, 1952.
12. Bourne, Geoffrey: Mitral valve disease in the light of surgical treatment, Brit. M. J. 1:896, 1952.
13. Wilcox, L. D., and Grace, A. J.: Barriers to surgery in mitral stenosis, Canad. M. A. J. 64:218, 1951.
14. Bailey, Charles P., Bolton, Houck E., and Redondo-Ramirez, Hector P.: Surgery of the mitral valve, S. Clin. North America, Dec. 1952, pp. 1807-1818.
15. Surgery of mitral stenosis (European Congress of Cardiology). Lancet 2:575, 1952.

BONY METASTASES AND SIMULATING LESIONS IN ADULT MALES

CHARLES P. ODERR, M. D.†
NEW ORLEANS

LOCALIZATION OF METASTASES IN GENERAL

The localization of a metastases depends on the *site* and *type* of the primary tumor.

The *site* of the primary will determine which of the three categories of capillary filters the tumor emboli will first encounter: (a) portal capillary filter in the liver, (b) pulmonary capillary filter, or (c) systemic capillary filters—chiefly bone, kidney, liver and brain.

The *type* of the primary tumor will determine whether the mechanical barrier of the first encountered capillary bed will be an effective filter. This is a function of the size of the metastatic embolus. Tumors of the type which have either connective tissue stroma or intercellular ground substance are apt to give off multiple-cell emboli because of the close union of the tumor cells. Multiple-cell size emboli will be effectively filtered out by capillary networks. On the other hand, tumors of the type which have no connective tissue stroma or intercellular ground substance have no close union of cells, and therefore, give off single cell emboli. These are not readily filtered out due to their small size, and they pass quickly into all parts of the body.[5] In this case the barrier to rapid establishment of generalized disease is biological. That is to say, that most of the emboli do not survive because of the unfavorable soil in which they become lodged. Favorable soil is homologous tissue, usually other parts of the same organ but sometimes similar tissue in other parts of the body (reticuloendothelial system). In homologous tissue single cell emboli may survive; whereas, they would not survive in other tissue. Later, when the disease is more advanced and the patient's resistance is weakened, this biological barrier is overcome; the metastases survive and grow in all tissues. The tumors of this type, i. e., having no close union of cells, are the anaplastic carcinomas, multiple myelomas, lymphosarcomas, leukemic neoplasms, and Ewings' sarcoma of bone marrow.[5]

We have discussed hematogenous metastases in general in order to show the relation of bone, as a metastatic filter, to metastatic filters in general. In those instances when the tumor emboli first enter a lymphatic vessel rather than a blood vessel, the lymph node barrier is interposed before the tumor cells reach the blood vascular system. Thereafter, they encounter *first* the pulmonary capillary bed. Thus,

* Presented at the Seventy-third Annual Meeting of the Louisiana State Medical Society, May 9, 1953, in New Orleans.

† From the Veterans Administration Hospital, New Orleans, Louisiana.

they form a special subgroup (from the hematogenous point of view) belonging to the second or pulmonary capillary filter category.

FREQUENCY OF BONY METASTASES

The special importance of skeletal metastases does not arise from their over-all frequency since this is only about 12 percent. However, in those tumors in which the systemic capillary beds are the primary filters, or in instances in which the tissue of origin is homologous with bone marrow tissues, the frequency is much higher, varying up to 100 percent.

The frequency with which a skeletal metastasis from a given organ is encountered is, of course, also a function of the frequency with which tumors occur in that organ. Thus, lung and prostatic carcinoma account for most of the skeletal metastases in man; whereas, breast accounts for most of the skeletal metastases in women.[1]

The chances of a given tumor having metastases is increased by the vascularity of the organ. Thus thyroid carcinoma is more apt to produce multiple hematogenous metastases than gastric carcinoma.

TYPES OF CHANGE IN RADIOGRAPHIC APPEARANCE OF THE BONE

From the radiographic point of view, there are two principal categories of skeletal change from metastases: osteoclastic, by action of osteoclasts; and osteoplastic, by action of osteoblasts.[4] For some cases, notably leukemic bone involvement,[6] it has been shown that the sclerosis does not follow directly but only after replacement of tumor cells by fibrosis. Except for an occasional breast tumor, lesions in females are almost entirely osteoclastic. In males, there is a more even balance between the two categories due to the large group of prostatic carcinoma, which are osteoplastic. A third category of metastatic lesions, from a radiographic viewpoint, should be made; namely, those that resemble primary bone tumors. This similarity of appearance is due to: (a) their peripheral location in the bone, which is contrary to the usual central location for metastatic foci (probably a function of statistical probability); and (b) the occurrence of periosteal proliferation and soap bubble effects, which mimic certain primary tumors.

CLINICAL—RADIOGRAPHIC CORRELATION

An important feature of skeletal metastases from a clinical standpoint is the early demonstrability of the lesions. This of course is also true of lung metastases, but is not true of the liver, kidney, brain, and internal lymph node metastases. However, not all skeletal metastases are visible early or easily. Spongy bone structure, which captures a high percentage of metastatic emboli lodging in bone due to its relatively greater vascular bed, will not show metastases early. According to Borak,[2] a destroyed area in spongy bone is not visible until about half of the thickness of bone, in the axis of the central ray, is destroyed. Diffuse lesions in spongy bone may never be visible except as generalized osteoporosis. On the other hand, any destruction of compact bone or any change in contour of a bone, especially partial collapse of a vertebra, is demonstrable immediately. Thus, in one case there may be clinical symptoms without visible bone lesions, while in the other there may be radiographic findings before clinical symptoms are manifest.

SPECIFIC SKELETAL LOCALIZATIONS

With regard to specific bones involved, it is not surprising that 80 per cent of cases with bone metastases will show spine lesions when we consider the great amount of vascular bed represented in the vertebrae. The intervertebral disc is not affected, thus providing a helpful diagnostic point in doubtful cases. The other major sites are femurs, ribs, sternum, pelvis, skull and shoulder girdle. The inherent difficulties in radiography of the base of the skull and sternum may often be responsible for missing lesions in these areas.

DIFFERENTIAL STUDY IN 184 CASES

A group of 184 cases of metastatic bone disease and simulating lesions was assembled at the Veterans Hospital, New Orleans, representing a period of about

six years. Representative examples of the same anatomical part from different cases were placed in juxtaposition for exhibit and study. The similarity of lesions of different etiology was striking and emphasizes the need to consider all diagnostic means, including biopsy, before arriving at conclusions. The cases were also grouped according to three major age categories: (I) under 40 years, (II) 40 to 60 years, (III) over 60 years. Subdivisions of these were: (A) cases showing little or no bone reaction, (B) those showing areas of increased bone density, resulting in six categories in all. Differential diagnosis as encountered in the six groups was as follows:

I. Under 40 years.
   A. Little or No Bone Reaction.
      Sarcoma: from all bone elements, lymphatic and reticuloendothelial systems.
      Carcinoma: a few from upper and lower respiratory tracts.
      Infections: tuberculosis
      Other: eosinophilic granuloma, chondroma, cysts, fibrous dysplasia, hyperparathyroidism.
   B. Areas of Increased Bone Density.
      Sarcoma: from osteoblasts.
      Carcinoma: from paranasal sinuses.
      Infections: osteomyelitis; bone abscess.
      Other: osteitis condensans, osteopoikilosis, Hodgkin's disease (Multiple poorly defined areas of medullary involvement become more sclerotic after x-ray therapy).

II. 40 to 60 years.
   A. Little or No Bone Reaction.
      Sarcoma: from connective tissue, muscle, cartilage, and osteoblasts.
      Carcinoma: from upper and lower respiratory, gastrointestinal, and urinary tracts.
      Infections: tuberculosis.
      Other: multiple myeloma (alkaline phosphatase not elevated; reversal of A/G ratio; confirmation by sternal puncture); gout; neurofibromatosis.
   B. Areas of Increased Bone Density.
      Sarcoma: from osteoblasts; associated with Paget's disease.
      Carcinoma: from prostate and upper respiratory tract. Any carcinoma may show a high alkaline phosphatase.
      Infections: brucellosis; tuberculosis; coccidioidomycosis.
      Other: plasma cell myeloma; old medullary infarction; Paget's and Paget's with multiple myeloma.

III. Over 60 years.
   A. Little or No Bone Reaction.
      Sarcoma: from osteoblasts.
      Carcinoma: similar to 40-60 group but frequency less.
      Infections: none in this group.
      Other: multiple myeloma; compression of body of vertebra, associated with senile osteoporosis. (This osteoporosis is due to **decreased** rate of osteoblastic activity in laying down bone matrix protein, probably due to endocrine deficiencies. With continued **normal** rate of osteoclastic activity there is a net loss of bone, and therefore, of calcium density. Diagnosis by exclusion of other specific causes of collapse is often necessary).
   B. Areas of Increased Bone Density.
      Sarcoma: none in this group.
      Carcinoma: prostate (usually nonreticular sclerosis, but occasionally lytic lesions; high acid phosphatase in serum; usually good response to estrogen therapy)
      Infections: none in this group.
      Other: Paget's disease (coarse trabecular structure is most common type, but cystic and non-reticular sclerotic lesions also occur; cortex thickened; often involves cranium and the hypertrophic spurs of an associated osteoarthritis; usually high alkaline phosphatase; often shows hyperplastic deposits about dental roots; bone softening occurs).

Although not encountered as a diagnostic problem in this series, mention should be made of (idiopathic) myelosclerosis. Characteristically, this is generalized, in contradistinction to most cases of malignancy and leukemia. The spleen shows marked compensatory enlargement in these cases and should not be removed.[6]

There are, of course, other tumors and diseases not encountered in this series, especially those peculiar to females and children. While it may be true that some specific lesions tend to produce individualistic roentgen findings, for the most part reliance must be placed on age, sex, distribution, the presence or absence of bone production associated with the bone destruction, and the correlation with significant clinical data.

CONCLUSION

The incidence of early skeletal metastases is determined partly by whether or not the emboli must pass through other capillary or lymphatic filters before reaching the bone. It is also determined in part by whether or not the primary tumor arises from cells which are homologous with any of the bone or bone marrow elements.

The incidence of skeletal metastases late in the course of the disease, when the first encountered filter and the homologous tissue barriers have broken down, is determined by the relative effectiveness of the several existing defense mechanisms.

Infectious and metabolic diseases may simulate metastases, but a careful consideration of all data will usually place the lesion in the correct major category. If the specific diagnosis depends upon elements of *microscopic* size, it is beyond the natural limitation of roentgenology.

REFERENCES

1. Abrams, H. L., et. al.: Metastases in carcinoma. Cancer, 3:74 (January) 1950.

2. Borak, J.: Relationship between the clinical and roentgenological findings in bone metastases. Surg., Gynec. Obstet. 75: 590. (November) 1942.

3. Galding, F. Campbell: Metastatic tumors of bone, diagnostic aspects. J. Faculty of Radiologists. 1:245 (April) 1950.

4. Shackman, R.: et. al.: Occult bone metastases. British J. Surg., 35:385 (April) 1948.

5. Schinz, Baensch, Friedl and Uehlinger: Roentgen Diagnosis, Grune and Stratton, II. 1952.

6. Windholz, Mark and Foster, S. E.: Bone sclerosis in leukemia. Amer. J. Roentgenol. 61:74 (January) 1949.

—o—

ECTODERMOSIS EROSIVA PLURIORIFICIALIS (STEVENS-JOHNSON SYNDROME) TREATED WITH CORTISONE *

MAHDI MURTADHA, M. B., CH. B.**
GLEN A. RAMSDELL, M. D.
MAXWELL C. SNIDER, M. D.

The acute febrile disorder characterized by pleomorphic inflammatory lesions of the skin and mucous membranes (Stevens-Johnson syndrome)[1] has heretofore been quite resistant to treatment. Several recent reports[2-4] have described dramatic responses to ACTH and cortisone. An equally gratifying experience with cortisone is herein reported.

CASE REPORT

E. B., a white woman, aged 61 years, was admitted to Binghamton City Hospital, on Dec. 30, 1950, with the chief complaint of a sore mouth and a cutaneous rash of three days' duration. There was no personal or family history of allergy or cutaneous eruptions. The present illness had begun on December 15, with chills, fever, and cough for which she had consulted a physician on December 23, and again on December 26; he gave her 300,000 units of penicillin intramuscularly on each occasion. Nevertheless, her eyes became inflamed, her mouth and throat became so sore she could scarcely swallow and an eruption appeared on her face, trunk, and extremities.

On admission to the hospital she appeared acutely ill; her temperature was 103° F., pulse rate 120, respirations 28, and blood pressure 140 mm./Hg. systolic and 100 mm./Hg. diastolic. In addition to acute bilateral conjunctivitis, the lips and entire buccal mucosa were acutely inflamed and studded with superficial ulcers of variable size (Fig. 1). Purpuric vesicles of pin-

Fig. 1.—Photograph of patient on admission showing cutaneous lesions in Stevens-Johnson disease.

head dimensions covered the malar area, the trunk, and the extremities. Patchy pneumonitis in the right lower lobe was demonstrated by physical and roentgenographic examinations.

Examination of the blood revealed a red blood

*From the Department of Medicine, Binghamton City Hospital, Binghamton, New York.
**Presently a Fellow in Internal Medicine, Alton Ochsner Medical Foundation, New Orleans.

cell count of 4,100,000 per cu. mm., hemoglobin 13 Gm., white blood cell count 8,500 per cu. mm. (stab 9, polymorphonuclear segmented cells 79, eosinophils 3, and lymphocytes 9). Sedimentation rate was 25 mm. (Westergren) and Wassermann negative. Urinalysis revealed 25 to 30 white blood cells per high power field, and no growth on culture. Febrile agglutinins were negative. Smears from the mouth and conjunctiva were negative on culture. Sputum cultures grew methemaglobin producing streptococci and nonhemolytic *Staphylococcus aureus*. The bone marrow showed hyperactivity with the cellular elements present in the normal ratio. The electrocardiogram showed no abnormalities.

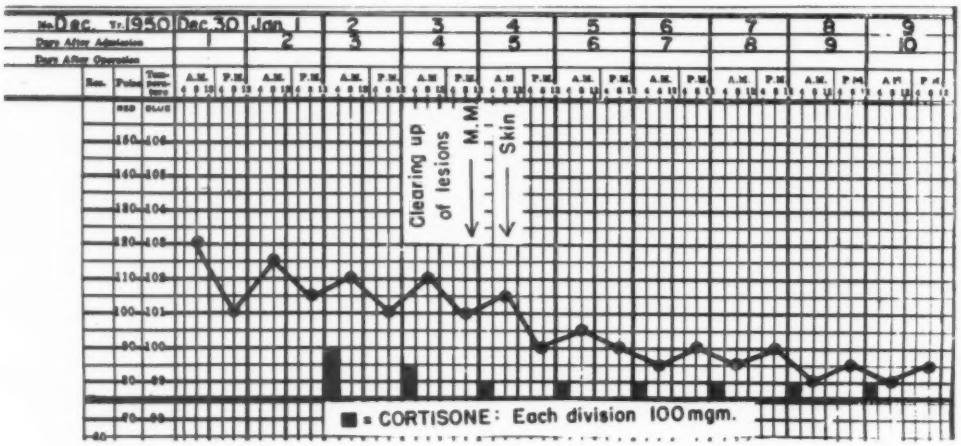

Fig. 2—Chart showing clinical course of the disease.

Clinical Course—Since aureomycin and benadryl in the usual dosage for three days had no effect, the intramuscular injection of cortisone in divided doses was begun on January 2: 300 mg. the first day, 200 mg. the next, and 100 mg. thereafter for six days. By January 4, when the patient had received 500 mg. of cortisone great improvement was obvious. The ulcerations and sloughs over the tonsils and pharynx had completely disappeared and the lesions over the buccal mucosa, tongue, and roof of the mouth had partially healed. Dysphagia had vanished. The rash as a whole was fading out and desquamating. The conjunctivitis had subsided except for one or two small areas. On January 5, the rectal temperature, which had been spiking remittently, dropped to 100° F. and thereafter remained normal; administration of aureomycin was discontinued. On January 7, the oral lesions had healed except for a small area over the hard palate. The conjunctivitis had also disappeared as did the rash several days later. By January 11, the patient was virtually without complaints, only a small crusting area remaining on the lower lip. Administration of cortisone, therefore, was discontinued on this date, the total dosage having been 1.1 Gm. over a period of nine days (Fig. 2). The following day the patient was discharged in good condition (Fig. 3).

Fig. 3—Photograph of patient on discharge showing healed lesions following administration of 1.1 Gm. cortisone.

SUMMARY

A severe case of ectodermosis erosiva pluriorificialis (Stevens-Johnson syndrome) associated with bronchopneumonia, successfully treated with cortisone, in a white woman aged 61 years, is presented.

Aureomycin and benadryl were given for three days without improvement. Forty-eight hours after institution of cortisone therapy pronounced improvement in the lesions was observed. Recovery was complete after ingestion of 1.1 Gm. of cortisone during a period of nine days.

REFERENCES

1. Stevens, A. M. and Johnson, F. C.: A new eruptive fever associated with stomatitis and ophthalmia: report of two cases in children, Am. J. Dis. Child. 24:526, 1922.
2. Wammock, V. S., Biederman, A. A. and Jordan R. S.: Erythema multiforme exudativum (Stevens-Johnson syndrome), J. A. M. A. 147:637, 1951.
3. Agostas, W. N., Reeves, Nathan, Shanks, E. D., Jr. and Sydenstricker, V. P.: Erythema multiforme bullosum (Stevens-Johnson syndrome), New England J. Med. 246:217, 1952.
4. Finn, J. J., Jr. and Mac Gillivray, D. C.: Erythema multiforme exudativum (Stevens-Johnson syndrome), Bull. New England Med. Center 14:28 (Feb.) 1952.

—o—

CHANNEL ULCER *

GEORGE E. WELCH, M. D.
JOSEPH MARINO, M. D.
CHARLES O'D. LILLY, M. D.
DONOVAN C. BROWNE, M. D.
NEW ORLEANS

INTRODUCTION

Peptic ulcerations of the pyloric channel present a most interesting problem, because of their variable clinical symptomatology, frequency of obstruction, gastritis, hypoacidity, weight loss, and radiological features in the absence of demonstrable craters, and the reported low incidence of malignancy despite the anatomical gastric location.[1] Recently, Texter[1] reviewed 55 cases delineating the clinical and radiological features. The present review concerns a corroboration of these features with emphasis on the clinical and radiographic triad and presentation of our experience with 21 cases.

DEFINITION

Radiologically the channel is considered to be that portion which is produced by a localized thickening of the longitudinal and circular layers of the propia muscle. Because of the blending of the muscle fibers into the adjacent antral wall it is felt that a rigid, exacting length cannot be stated. However, it is felt that it ranges from 0.5 cm. to 1 cm. Therefore, any length above 1 cm. should be regarded with suspicion.[3]

CLINICAL MATERIAL

The symptomatology in 21 cases with demonstrable channel craters is outlined below (Table 1). The symptomatic triad[4] consists of: (1) Short stormy episodes of nausea and vomiting due to intermittent pyloric obstruction; (2) pain of variable character, usually relieved by food, but followed by abdominal distention and bloating sensation one to three hours after meals; and (3) weight loss of more than 10 pounds. The mean free and total gastric acidity for the group following an Ewald test meal was 30 degrees and 42 degrees, respectively.

TABLE I
SYMPTOMS (21 CASES)

SYMPTOMS	NUMBER	PER CENT
Nausea	18	85.5
Vomiting	13	62
Epigastric pain relieved by food and followed by abdominal distention 1 to 3 hours after meals	13	62
Pain, unrelated to food	5	24
Night pain	5	24
No pain	2	9.5
Weight loss of more than 10 pounds	5	24
Bleeding	2	9.5

Radiographic evidence of a crater in the pyloric canal is diagnostic of ulceration. This was seen in all 21 cases. One of the lesions was found to be malignant. Antral gastritis or spasm occurred in 17 instances. Five cases showed abnormality of the pyloric ring and the duodenal cap was deformed in 11 cases. The ulcers were confirmed surgically in 5 cases. Significant gastric retention, although an important finding, was present in only 3 of the 21 cases in this series. In the absence of a crater, the radiological triad[2] of antral gastritis or spasm, deformity of the duodenal cap, distortion or lengthening of the pyloric canal may suggest the entity.

While the incidence of cancer of the stomach is about 5 per cent of all gastric

* Presented at Meeting of the Orleans Parish Medical Society, December 14, 1953.

From the Gastroenterological Division of the Departments of Medicine and Radiology, Browne-McHardy Clinic, and Tulane University School of Medicine, New Orleans, Louisiana.

ulcerations [5] it has been reported unusual to encounter malignancy in the pyloric channel. [1] In this study it was present once representing an incidence of about 5 per cent. This suggests that the incidence of malignant ulceration in this site is comparable to that elsewhere in the stomach.

Channel ulcers are gastric ulcers, and they should be managed as such. If they do not heal or do not show evidence of marked healing within a period of thirty to forty days on a strict medical program, we feel they should be removed surgically.

SUMMARY AND CONCLUSIONS

1. Twenty-one cases of pyloric channel ulcers have been reviewed.

2. The clinical and radiological features were emphasized.

3. These lesions are located in the stomach and should be managed surgically if they are not healed or do not show evidence of marked healing after a period of thirty to forty days of intensive medical management.

REFERENCCES

1. Texter, E. C., Baylin, G. J., Ruffin, J. M., and Legerton, C. W.: Pyloric channel ulcer, Gastroenterology, 24:319, 1953.

2. Smedal, M. I.: Pyloric ulcers, Radiology, 39:200, 1942.

3. Doub, H. P.: The differential diagnosis of pyloric and prepyloric ulceration, Am. J. Roentgenol., 48:826, 1940.

4. Eusterman, G. B.: In discussion on Butsch, W. L.: Ulcers of the pyloric ring, Proc. Staff Meet., Mayo Clin., 10:436, 1935.

5. Browne, D. C., Mitchell, R. E., Jr., McHardy, G., Welch, G. E.: Evaluation of surgical intervention in gastric ulcers, submitted for publication.

The Journal of the Louisiana State Medical Society

Established 1844

Published by The Journal of the Louisiana State Medical Society, Inc. under the jurisdiction of the following named Journal Committee:

Philip H. Jones, M. D., Ex-Officio
E. L. Leckert, M. D., Chairman
C. M. Horton, M. D., Vice-Chairman
Sam Hobson, M. D., Secretary
Edwin H. Lawson, M. D.
J. E. Knighton, M. D.

EDITORIAL STAFF

Philip H. Jones, M. D. ..... *Editor*

COLLABORATORS—COUNCILORS

H. Ashton Thomas, M. D.
Joseph S. Kopfler, M. D.
Guy R. Jones, M. D.
Paul D. Abramson, M. D.
C. Prentice Gray, Jr., M. D.
Arthur D. Long, M. D.
J. W. Faulk, M. D.
H. H. Hardy, M. D.

C. Grenes Cole, M. D. ..... *General Manager*
1430 Tulane Avenue

SUBSCRIPTION TERMS: $4.00 per year in advance, postage paid, for the United States; $4.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

The Journal *does not hold itself responsible for statements made by any contributor.*

SAFETY AND PROSPECTIVE BENEFITS FROM POLIO VACCINE

The National Foundation for Infantile Paralysis has planned, and is about to carry through, massive field trials of a vaccine designed for immunization against polio. This vaccine was developed by Dr. Jonas E. Salk of the University of Pittsburgh and represents an achievement whose value is difficult to calculate. It is the result of the cumulative effort in this research on the part of some of the best investigators in American medicine.

The vaccine is a killed culture of the three types of polio virus, grown on a nutrient fluid, containing minced, monkey kidney tissue. The virus is killed by USP formaldehyde, in which the final concentration in the solution is 1:4000. After six to twelve days of the action of the formaldehyde, it is neutralized by the addition of sodium bisulphite. Following this, a system of tests has been set up for the determination of the suitability of the vaccine for use. These tests are for safety and sterility and involve tissue culture, inoculation of monkeys, mice, rabbits, and guinea pigs.

In preparation for the production of vaccine to be used on a national scale each batch of vaccine is to be tested by three laboratories, each of which is carrying out the complicated procedures for safety and sterility. The laboratories are that of the manufacturer, secondly, the Virus Research Laboratory of the University of Pittsburgh, directed by Dr. Salk, and the Laboratory of Biologics Control of the National Institute of Health. Each batch must receive unconditional safety approval from all three before it is released for packaging and shipping to the field trial areas.

These arrangements have been reviewed and concurred in by the members of the special Advisory Committee on Active Immunization of the National Foundation for Infantile Paralysis. The Chairman of this Committee is Dr. Thomas N. Rivers, Director of the Hospital, of the Rockerfeller Institute for Medical Research, and one of the leading virologists of the nation. The other six members are equally well qualified in closely associated fields.

Seldom has any material for immunization been prepared with as much background of research and with as much care for protection as this one.

Previous to the planning of field trials, the vaccine had been given to about 8000 children and adults. Elaborate investigations have shown that production of antibodies in individuals who did not possess them previously was initiated by the vaccine, and that among those who had anti-

bodies before injection, an increased amount of antibody was shown. It had previously been shown by elaborate researches that the three types of polio virus follow the established laws of immunity. Reasonably, therefore, Dr. Salk and his associates anticipate that antibody production would be expected to be accompanied by protection against tissue invasion and paralysis. It is this hoped for protection against paralysis which field trials will show, and not, as has been suggested, "the safety of the vaccine."

At this point in the preparations which were going on over the nation in forty-four states, a radio authority announced, on April 4, that the vaccine "may be a killer." The basis for such a comment was stated to be that in the process of testing for safety and sterility some of the batches of vaccine were found to contain live virus. This comment on a national broadcast, even though it came from one who is not infallible, has occasioned concern on the part of the boards of health, on the part of those sponsoring the field trials, and also, on the part of the parents of the children who might receive the vaccine. Explanations from Dr. Salk and those associated with him have been clear and reassuring. As might have been expected, the explanation is that a system of controls was set up to insure proper testing of each batch of vaccine, and this system was doing just what it was set up to do, that is, eliminating what was not suitable. The course of events is actually a compliment to the care, forethought and scientific precision of those in charge of the whole endeavor.

When the field trials were proposed, a committee of the Louisiana State Medical Society, in cooperation with the Louisiana State Board of Health, approved the proposed use of the vaccine. When the radio comments produced an acute concern about the safety of the procedure, this Committee issued a statement from the Louisiana State Medical Society, stating in part:

"We still believe that in view of all information known about this vaccine that it holds out good prospects for benefits to be derived from its use, and if our people desire it on a voluntary basis they should not be denied the privilege of receiving it."

An additional phase of the problem has been commented on by Dr. Salk himself in a recent address before the New Orleans Graduate Medical Assembly, as follows:

"Because of the fact that virus for vaccine is propagated in cultures of monkey kidney tissue, some have wondered whether or not immunization with such vaccines may have some damaging effect upon the kidneys. Although, on a priori grounds, there is little reason to believe that this might occur, this question has been the subject of continued study, in a variety of ways, and there are no indications thus far of any harmful effect upon the kidney, either in experimental animals or in man."

It is understood, at the time of this summary, many hundreds of individuals had been examined without any evidence of kidney involvement.

It seems clear from the facts in the situation that the vaccine offers the best prospect of protection against the paralytic phase of polio that has existed up to now. In this, as in many other instances, medical problems should be decided by physicians. It is hoped that the field trials will proceed as planned and that the results will be illuminating and helpful.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

PHILIP H. JONES, M. D.
President
1953 - 1954

Dr. Philip H. Jones, our President during the current year, has been most energetic and ever alert to the needs of our Society. His insight concerning medical matters has been keen and he has been ever watchful for the possibility of any problems which might be detrimental, in any way, to organized medicine and our Society. We have been extremely fortunate in having a man like Dr. Jones, with his unsurpassed capabilities and breadth of vision, combined with his willingness and desire to serve, as our leader for the past year. It has, indeed, been an exceptionally busy year, filled with many problems necessitating, at times, the most mature and considered action. In solving these problems, he has always kept in mind what was best for the public and for our Society, and in doing so has merited the respect and confidence of our members. Dr. Jones has conducted the affairs of the Society on a high and dignified plane, always creating interest and eliciting attention and cooperation from his fellow members. He has always had the courage of his conviction, but on the other hand, has always been considerate of the opinions of others and has gladly welcomed the advice and counsel of his colleagues.

Dr. Jones has energetically and with his usual ability and great resourcefulness, most ably represented the profession at many state and national conventions and conferences. He has been extremely active in participating in the district and parish society meetings, along with many hospital staff meetings. Wherever duty called, Dr. Jones has responded and unequivocally defended organized medicine and the profession in a manner which always left behind that feeling of good public relations and fellowship between our profession and the lay public, for which we are always striving.

Dr. Jones was born in Jackson, Louisiana, and in 1907 his parents moved to Baton Rouge, where his father, Dr. Philip Huff Jones enjoyed a large medical practice. His mother, Mrs. Annabelle Smith Jones, is still living in Baton Rouge at the age of 92 years.

He received his early education in the Baton Rouge Public Schools, and in 1916 received his B.A. Degree from the Louisiana State University. His M. D. degree was received from Tulane University in 1920, being the third generation of his family to graduate in Medicine from Tu-

lane; his grandfather graduating in 1852 and his father in 1878.

His internship was started at Charity Hospital in 1920 and four months later his training was interrupted to accept a Rhodes Scholarship and he earned his Ph.D. Degree from Oxford University in 1924. His studies, while abroad, included the Clinics of Vienna, a course in dermatology in the Hospital of St. Louis, Paris, and cardiology in the London Hospital and the London Heart Hospital. He returned to New Orleans, where he completed his internship in 1925, and served as House Physician at Charity Hospital from 1926 until July, 1928. He commenced private practice, specializing in internal medicine, in 1929.

Dr. Jones has served on the teaching staff of Tulane Medical School, as full time instructor, part time instructor; as assistant professor of clinical medicine and associate professor of internal medicine.

Dr. Jones, while an instructor in the School of Medicine of Tulane University in 1934, the year of its One Hundredth Anniversary, was signally honored by being selected by the graduating class as outstanding among the active faculty because of his solid scholarship, intellectual precision and professional reliability.

He has taken quite an active part in organized medicine having held important offices in the Orleans Parish Medical Society, and served with distinction and honor as its President in 1945. He has likewise served the Louisiana State Medical Society in many capacities, and was elected its President in 1952, to be installed at the 1953 Annual Meeting.

He is a member of the American and Southern Medical Associations, is a Fellow of the American College of Physicians, and was certified by the American Board of Internal Medicine in July, 1938. He is a member of the Nu Sigma Nu and A.O.A. Fraternities.

He was in the S.A.T.C. at Tulane University during the first World War, and in World War II he served as examining physician for the local Boards; also as a member of Medical Advisory Board No. 1, and as Assistant Chief of Medical Service in the emergency unit formed at Charity Hospital in New Orleans.

Dr. Jones has contributed many scientific papers to our local and national journals, and has been the author of a series of chapters on infectious diseases in one of our reference books on medicine.

He is a member of the visiting staffs of the Southern Baptist Hospital, having served as its President. He is serving as a member of the Board of Administrators of the Charity Hospital at the present time.

He takes quite an interest in civic and community problems, having served just recently as Chairman of the Medical Committee of the New Orleans Chamber of Commerce, in which position he did an outstanding job. He is also a member of the Louisiana Health Council, contributing his talents and time to this worthwhile organization.

Dr. Jones has served as Editor of the Journal of the Louisiana State Medical Society since 1948, and has rendered efficient service, and the members of our Society owe him a great debt of gratitude for his untiring interest as Editor of the Journal.

Dr. Jones has been very active in our recent fight against the licensing of Chiropractic in our state, and has been working unceasingly in promoting our educational program of the public against this cult. If the medical profession, as a whole, would do just half as much as Dr. Jones is doing to defeat the chiropractic menace, we would have no fear of the results to be obtained in Baton Rouge at the coming session of the Legislature.

The members of our Society have been very fortunate, indeed, in having Dr. Jones as their President during the past year, and it is comforting to know that we will still be permitted to enjoy his mature judgment and wise counsel as a member of the Executive Committee. It is our sincere hope that he will continue his keen

interest in organized medicine throughout the coming years. Please be assured, Dr. Jones, that we appreciate your wonderful service to our Society.

REPORT OF PRESIDENT

The past year has been one of many activities and complex responsibilities for the officers of the Louisiana State Medical Society, the Executive Committee, and for the standing and special committees.

Organizational Activities

The Society was represented at the meeting of the A. M. A., in June 1953, in New York. At this meeting, appearances were made before the Committee of the A. M. A. on Medical Service and Hospitals, in regard to your protest on the requisites for an approved internship. The effect of these representations made along with those of three other states was to neutralize the regulation for the present, and to procure reconsideration of the whole field. For the present, and possibly for the future, an embarrassing situation has been avoided which would affect all the hospitals of the state except two. Also, at this meeting, there were attendances at the sessions of the House of Delegates, and at sessions devoted to the state presidents and secretaries. At the latter, matters of organization detail were discussed, and much was learned which will be of benefit to the Society.

A similar meeting of the A. M. A., in St. Louis, was attended. Sessions of the House of Delegates were followed, and conferences of state officers were found beneficial.

National Conferences

Dr. Robyn Hardy was sent as representative to the regional conference for veterans' affairs, sponsored by the A. M. A. at Dallas. In this conference an effort was made to inform the several state societies of the status of veterans' hospitalization of patients with nonservice connected disabilities. Its object was to coordinate the planning in the several states to meet this problem. The situation is urgent because of the growing number of veterans, the probable increase in their need for hospitalization, and our knowledge of the fact that with ease a greatly expanded hospital program on the part of the Veterans Administration could be suddenly authorized to care for families as well as veterans. In such a process, State Medicine could become established.

Dr. J. P. Sanders represented the Society at the Rural Health Conference in Dallas. The influence and beneficial effect of this Conference has been increasing year by year, and its activities are ultimately going to be helpful to rural areas. Under its stimulus, progressive improvement in rural medical care is being noted.

Our immediate Past President, Dr. Will Barker, attended the Rural School Health Conference near Chicago. His report shows the value of the activity of this group, and its benefits in promoting rural health.

Dr. Robyn Hardy attended the Public Relations Conference of the A. M. A. in Chicago. His report is another indication of the need for our continued support and concern in this field.

The American Medical Education Foundation met in Chicago last February, and the Society was ably represented by Dr. Edgar Hull. This Foundation is organized medicine's effort to supply funds for medical schools and to avoid federal government subsidy.

Activities Within The State

The President and Secretary attended the annual meeting of the Louisiana Health Council in Alexandria and found the sessions stimulating and productive of better relations for the State Society. The Louisiana Health Council is the most valuable forum in the state on health matters, outside of our own Society, and it seems most desirable that a close relationship be encouraged and maintained. The public in many areas is in need of a means by which medical aims could be interpreted and realized within the community. The Louisiana Health Council gives promise of great value in this field.

The sessions of the Legislative Commission on Workmen's Compensation Law

were attended, with Dr. Roy Harrison as spokesman for the Society. The position of Organized Medicine in this controversial field was adequately explained and admirably upheld. The position taken, in substance, was that the Civil Courts were preferable to a commission. It was also explained that the position of the doctor in the case should receive greater financial protection, but otherwise, the position of the doctor in lawsuits for compensation should be unchanged.

The officers attended the meeting of the Louisiana Academy of General Practice, in Alexandria, last fall. The meeting was well attended and enthusiastic. This group now, as in the past, is upholding the best traditions of medicine in Louisiana and enjoys the continued cooperation of the Louisiana State Medical Society.

Meetings of various parish and district societies have been attended when possible, and in eight of these opportunity was additionally offered to meet and speak to legislators against the bill to license chiropractors.

The Executive Committee was called into session in June to consider and act upon current matters and those referred from the House of Delegates. It was also called on December 12, 1953, to consider the chiropractor situation and related problems. At this meeting, authorization was granted to the Committee on Public Policy and Legislation to employ a firm of public relations consultants, Gould, Blieden & Manley, of Baton Rouge. It was also decided that donations should be requested from the membership to meet the necessities of the educational campaign against chiropractors, which has been and is currently being waged. The Committee was polled once over the telephone for a decision about supporting legislation to license physiotherapists, and to investigate chiropractic. The collected vote was against the endorsement of these projects.

Many of the standing and special committees have had more than the usual amount of work to do, and my sincere appreciation is expressed to them for their efforts in behalf of the Society. Of particular mention are the Committee on Public Policy and Legislation with Dr. Henry Jolly as Chairman; the Committee on Child Health, with Dr. Sims Chapman as Chairman; the special Committee on Mental Hospitals to work with the State Board of Institutions, with Dr. Edmund Connely as Chairman; the Committee on Veterans Affairs, with Dr. Pascal Danna as Chairman; the Committee on Public Health, with Dr. J. D. Martin, Chairman; the Cancer Committee, with Dr. Ambrose Storck, Chairman; and the Council on Medical Service and Public Relations, Dr. Robyn Hardy, Chairman.

Activity Against The Passage Of The Proposed Law To License

A considerable proportion of the activities of your Society's officers and many members has been absorbed in the past year in an effort to educate the public and secure legislative support against the Chiropractor Bill. This has been carried on in cooperation with the Committee on Public Policy and Legislation, Dr. Henry Jolly, Chairman, and it is evident that although the same problem has been presented every two years since 1922, our opposition is not adequately understood. It is also evident that the immunity we have enjoyed from the ill effects of this cult is more gravely threatened than previously. Accordingly, strenuous efforts have been, and will continue to be necessary over a period of years, to acquaint our membership, to educate the public, and to arouse public feeling in such a way as to provide adequate local enforcement of the Medical Practice Act.

It has been an honor and a pleasure to serve as President of the Louisiana State Medical Society. What has been accomplished was only made possible through the cooperative efforts of all concerned. I wish to thank particularly for their advice and support: Dr. C. Grenes Cole, Secretary-Treasurer; Dr. Will Barker, Dr. Henry Jolly, Dr. Roy Harrison, Dr. Robyn Hardy, and Dr. R. H. Riggs. The efficient office force, particularly Miss Annie Mae Shoemaker has contributed greatly to the success of our collective efforts.

RECOMMENDATIONS

The necessary activities of the Society are greater than its financial income. It is recommended consideration be given to increase in dues.

A recent decision of a Civil District Court in Louisiana has resulted in changing the effect of the statute of limitations for malpractice suits from one year to ten years, during which time a physician, dead or alive, can be sued for breach of contract. It is recommended that the House of Delegates endorse the efforts of our legal advisor, Mr. St. Clair Adams, to have a law passed which will return us to our previous status.

The multiplicity of insurance information blanks for which the fee is not paid by the company is a clerical hardship, and a tax on the time of the physician. It is recommended that a uniform insurance blank be endorsed by the House of Delegates, and that its establishment by legislative Act be sought.

The Society has had the services of public relations consultants (Messrs. Gould, Blieden & Manley) in the present campaign against chiropractors. This need may continue. It is recommended that the House of Delegates discuss the matter with a view to having a committee appointed to study the problem and report at the next meeting of the Society.

Because of the difficulties in securing accommodations at the place of the Annual Meeting, it is recommended that plans for meetings be made several years in advance.

The growing cost of malpractice insurance and the recently manifest tendency of juries to give greatly increased damages have produced a situation which affects every physician. It is recommended that the House of Delegates request constituent societies to devote one meeting each year to this phase of the practice of medicine.

The value of a School Health program has been demonstrated, and the productive part of each such project is the School Health Committee of the local society. It is recommended that the House of Delegates request each constituent society to appoint a School Health Committee from its membership, and that the whole Society cooperate with the School Health program.

The ultimate solution of the handling of the chiropractor problem is going to depend upon public support. It is recommended that the House of Delegates request the local societies to continue their special committees in this educational campaign.

PHILIP H. JONES, M. D.
President

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

WARNING

DON'T BE A SUCKER

LOUISIANA SAFETY ASSOCIATION wishes to advise all civic organizations and fraternal organizations in the State of Louisiana that two promoters are canvassing civic clubs in various towns for the purpose of selling safety materials under the guise of setting up safety campaigns in local cities in conjunction with the civic organizations.

The deal made to the civic organization is that 50% of the money goes to them to pay for their material and their time; and the other 50% goes to the civic organization for some charitable use. An operator has been in Marshall, Texas, and approaches have been made to several civic clubs in Shreveport.

Caddo Bossier Safety Council of Shreveport and Louisiana Safety Association, both chapters of the National Safety Council do not sanction or foster the exploitation of safety campaigns in communities.

It is requested that the members of your organization be advised of the solicitation program.

SOUTHEASTERN REGION MEETING OF THE AMERICAN COLLEGE OF PHYSICIANS

Dr. E. Dice Lineberry, of Birmingham, Governor of the American College of Physicians for Alabama, has scheduled a meeting of the Southeastern Region for October 15 and October 16, 1954, at the Edgewater Gulf Hotel, Edgewater Park, Mississippi. A special invitation is extended to members of the College and other physicians of Louisiana. Anyone wishing to appear on the program should contact Dr. Howard Holley, 620 South 20th Street, Birmingham, Alabama, who is the Chairman of the Program Committee.

ANNUAL MEETING AMERICAN MEDICAL ASSOCIATION

San Francisco, Calif.
June 21-25th, 1954.

The Missouri Pacific Lines offer through air-conditioned streamline service to San Francisco, with no change enroute. Daily convenient schedules with various sight-seeing attractions enroute.

Many of the famous National Parks are located in territories served by this route where stopovers can be made.

For further information—contact: Mr. J. T. Leze, General Agent or City Passenger Agents R. J. Conley and E. Mitchell, at RAymond 8244.

ANNUAL MEETING DIABETES ASSOCIATION OF LOUISIANA

The annual meeting of the Diabetes Association of Louisiana will be held with a luncheon during the State Medical meeting at a time and place to be designated on the first day of the meeting.

IT ISN'T SECOND CHILDHOOD— IT'S THE EFFECTS OF AGING ARTERIES

It isn't second childhood that oldsters go through—it's the effects of aging arteries.

But, one shouldn't let these effects distract one from the charm and wisdom underneath, according to Dr. John E. Eichenlaub, Urbana, Ill. A real understanding of the problem of why old people act differently helps one to enjoy the oldsters' company more, to keep feelings from being hurt, and to make oldsters happier and more content.

"You can't understand old people without understanding the changes in mind and personality that go with extreme age," Dr. Eichenlaub wrote in Today's Health magazine, published by the American Medical Association. "These changes don't always happen the same way or at the same age.

"A person is as old as his arteries, not as old as his years. Since the arteries that supply different parts of the brain wear out at different times and to different extents in different people, every person has his own way of showing age. But some changes almost always take place, and anyone who is really old, instead of in a sort of advanced middle age, is likely to have them."

REPORT CORTISONE AIDS IN TREATMENT OF BELL'S PALSY

Speedy recovery from Bell's palsy following the use of cortisone was reported in a recent (January 9) Journal of the American Medical Association. Bell's palsy is a common disorder of unknown origin which causes paralysis of the facial nerves and muscles. It usually takes many months to effect recovery. Little or no progress in its treatment has been reported until recently.

Two cases of immediate, complete recovery from the disorder were reported by Drs. William P. Robinson and B. F. Moss, Augusta, Ga. One case was that of a 13-year-old girl who recovered within two weeks after cortisone therapy was instituted. The second case was that of a 5-year-old boy who recovered within three weeks after treatment with cortisone was begun.

"The use of cortisone therapy in these two patients was followed by prompt recovery," the doctors wrote. "No patient suffering from Bell's palsy seen previously in our department over the last 15 years has recovered in less than several months.

"We are encouraged by the results of cortisone therapy in these two patients suffering from the early stages of Bell's palsy. Obviously, we cannot draw sweeping conclusions from observations so limited. We feel justified in the hope that others will test the use of cortisone in this disorder."

The doctors are associated with the department of psychiatry and neurology, Medical College of Georgia.

NEW PLASTIC PLOMBE FROM DENMARK COLLAPSES LUNG FOR TB TREATMENT

A new plastic filling material (plombe) developed in Denmark for partial lung collapse in treatment of pulmonary tuberculosis has been

made available to the medical profession in the United States by Lakeside Laboratories, Inc. here.

In plombage the chest is opened, part of the lung around the lesion is collapsed, and the filling material is left permanently in place. This promotes healing and prevents the spread of the lesion. The method has advantages over thoracoplasty, an operation in which ribs are removed and the entire lung collapses. Plombage provides selective collapse and avoids deformity and complications. The ribs are saved. As the blood supply comes from the ribs, plombage keeps intact the natural barriers against the spread of tuberculosis.

Called Polystan Plombe, this new type of high-molecular plastic is said to offer distinctive advantages over previously used materials. Bone and wax materials, for example, had to be abandoned because they caused foreign body reactions. Other plombes resembling "ping-pong" balls have been too mobile and inappropriate in size. The new Polystan Plombe is unaffected by body fluids and causes no foreign body reaction.

VOMITING IN CHILDREN MAY BE SIGN OF EMOTIONAL DISORDER

Vomiting may be the first and only indication of an emotional disorder in an infant or child, in the opinion of Dr. Paul C. Laybourne, Jr., Kansas City, Kan.

"The infant has only a few ways in which to express undue emotional tension," Dr. Laybourne wrote in a recent issue of the American Journal of Diseases of Children, published by the American Medical Association. "He can refuse food, cry excessively and vomit."

Much psychological vomiting in infants and children is the result of a disturbing atmosphere at home, Dr. Laybourne pointed out, stating:

"It is obvious in such cases that direct treatment of the baby or child is unnecessary. Psychological vomiting in infants is easily diagnosed by the simple expedient of hospitalizing them. Almost universally the vomiting stops with the removal of the baby from the disturbing environment of the home. This observation helps in making a differential diagnosis between organic and psychologic disease."

BOOK REVIEWS

Source-Book of Medical Terms; by E .C. Jaeger, Springfield, Illnois. Charles C. Thomas, 1953, pp. 145. Price $5.50.

This is a very useful book, especially for Freshmen and Junior medical students, for in these two years particularly they are inundated with new words. Knowledge of combining forms will greatly facilitate the acquisition of a medical vocabulary. If this book is used to go to sleep by and glanced at a few minutes each night, substantial help will be derived by the medical neophyte. The author has devised very good illustrations to show the ancient and modern meanings of medical terms.

In his introduction he states that he has found many inaccuracies in medical dictionaries and has corrected them. Alas! even Homer nods and it is regrettable that Dr. Jaeger himself has made some errors. Thus, in the very first entry he gives aasmos, Gr. asthma. Reference to Liddell and Scott's Greek Lexicon, his authority, indicates that aasmós, (aázō to breathe through the mouth) means a breathing out (p. 1) whereas asthma, atos, tó (áō to blow) means a short drawn breath, panting (p. 231). This incorrect derivation is repeated again on the same page by Dr. Jaeger although on p. 17 he gives asthma properly as from Gr. asthma. Again, on p. 137 he says: "The Latin word *trunco* means to *lop off, to cut off.*" *Trunco* is the first person present tense indicative and means *I* lop off, *I* cut off. *Truncare* is the infinitive form and means *to lop off.*

Nevertheless he has gathered much useful information and the book can be recommended to its designated audience; few medical students of today have had intimate contact with the ancient tongues.

VINCENT J. DERBES, M. D.

Hippocrates on Intercourse and Pregnancy; an English translation of On Semen and On the Development of the Child; by Tage U. H. Ellinger, New York, N. Y., Henry Schuman, Inc., 1952, pp 128. Price $2.50.

A first time English translation of a subject that should be of historical interest to all concerned.

I. L. ROBBINS, M. D.

Roentgenology in Obstetrics and Gynecology; by William Snow, M.D., Springfield Illinois, Charles C. Thomas, 1952. Pp. 363. Price $10.50.

This is the second edition of Dr. Snow's text on obstetric roentgenology in which there has been added the use of x-ray in gynecology also. There is excellent and complete coverage of pelvic architecture, fetal development, and the pathological changes which occur. The author has had wide experience with the problems of obstetrics and has devised a simple and effective method of x-ray pelvimetry.

Some obstetricians may be at variance with his views on the purely obstetric material which he has presented and possibly this detracts, to a certain

extent, from the text as a whole. However, the book is complete and well illustrated with a considerable number of excellent roentgenograms and can be recommended as a reference work for its fine radiographic presentation and interpretation.

GEORGE T. SCHNEIDER, M. D.

Clinical Neurology; by J. M. Nielsen, 3rd Edition, New York, N. Y., Paul B. Hoeber, 1951, pp. 709; price $10.00.

The third edition of Dr. Nielsen's text book continues to serve as a comprehensive guide to clinical neurology especially adapted to the needs of the medical student. The coverage is complete including all the clinically important disease entities. The text is well illustrated especially with photographs of pathologic specimens. The style is clear, and the author's use of personal case material helps to enliven the presentation of clinical data. The new edition keeps abreast of recent developments in the therapy of neurological disease. Especially helpful are the sections on the newer diagnostic techniques of angiography and electromyography.

A critical note may be offered in regard to the section on aphasia. The author, one of the country's leading contributors to the literature on this subject, acquaints the reader thoroughly with the clinical phenomena of aphasia, a topic often slighted in other texts. However, the reader may be lured into a rigid localization-bound approach to aphasia by the author's seemingly convincing but rather mechanistic viewpoint.

A. W. EPSTEIN, M. D.

Operating Room Technic; by St. Mary's Hospital, Rochester, Minnesota, 4th ed. Philadelphia, Pa., W. B. Saunders Co., 1952. Pp. 345. Price $6.50.

The Operating Room—Instructions for Nurses and Assistants, written in 1924, was revised for the third time in 1937. The present volume, the fourth edition, has been completely rewritten. Because of recent advances in surgery new procedures have been added and others revised.

The book outlines in detail the basic principles of aseptic technic and general principles of scientific management as practiced in the operating rooms of St. Mary's Hospital at Rochester, Minnesota. The stress is simplicity and uniformity of procedure for optimum skill. Restoration of the patient to health is the purpose, the climax, and the final end of the teams that function daily in the operating rooms through the world.

St. Mary's Hospital, operated by the Sisters of St. Francis of the Congregation of Our Lady of Lourdes, is a general hospital with a capacity of 850 beds and 60 bassinets. The patients are cared for in the departments of medicine, surgery, neurology, psychiatry, obstetrics, pediatrics and communicable diseases. The medical staff of the hospital is the staff of the Mayo Clinic.

Operative procedures are clearly and concisely outlined. The title of the operation, the definition of the operation, and the preparation and procedure in the operating room is given. There are eight chapters in the book, including general surgery with its sub-specialties, and orthopedic surgery, neurologic surgery, and procedures in urology and peroral endoscopy.

Development in the basic and advanced educational programs for nurses in the operating room are described. Diagrams of the position of the surgical team during various operations, illustrations of the patients in position for surgery, photographs of instrument arrangements, illustrations and measurements of linens have been included.

This book has a valuable fund of information for both the medical and nursing personnel. The volume should be of assistance to all who are interested in the field of operative surgery.

GEORGE W. HAGERMAN, M. D.

PUBLICATIONS RECEIVED

W. B. Saunders Co., Phila.: Manual of Clinical Mycology, by Norman F. Conant, Ph. D., David Tillerson Smith, M. D., Roger Denio Baker, M. D., Jasper Lamar Callaway, M. D., and Donald Stover Martin, M. D. (2nd. Edit.); Mayo Clinic Diet Manual, by The Committee on Dietetics of the Mayo Clinic (2nd. Edit.); Current Therapy 1954, Latest Approved Methods of Treatment for the Practicing Physician, edited by Howard F. Conn, M. D.

Simon and Schuster, N. Y.: A Doctor Talks to Women, by Samuel Raynor Meaker, M. D.

Charles C. Thomas, Publisher, Springfield, Ill.: Amebiasis, by Ernest Carroll Faust, Ph. D.; Menorrhalgia: Menstrual Distress, by William Bickers, M. D.